THE
LARYNGOSCOPE.

VOL. XLVI MAY, 1936. No. 5

THE OCULAR MANIFESTATIONS OF RHINOGENIC AND OTOGENIC INTRACRANIAL COMPLICATIONS.*†

DR. EDMUND B. SPAETH, Philadelphia.

This problem of the ocular manifestations of the intracranial complications of otitic and rhinologic pathology can be approached by several different routes: First, anatomic, that is, as to the source; second, intracranial pathology, and grouping of the foci of origin into one without considering their origin, but laying stress upon their pathways and results; and third, from the standpoint of their ophthalmic aspects or findings, both subjective as well as objective. Each would be of interest, but in each instance the purpose of the presentation would have to be different.

The rhinologist would favor the first; the neurologist, the second; and the ophthalmologist, the third. If this is correct, the third, must of necessity, be outstanding herein, though the first will be included. At the same time, with voluminous literature, which embraces everything from an exhaustive tome on otological complications to the recital of a single and not unusual case of Gradenigo's syndrome, it will be difficult, in fact impossible, to do more than sketch in some of the more important of these ophthalmic signs and symptoms.

*Read before the Philadelphia Laryngological Society, Philadelphia, Feb. 4, 1936.

†From the Peter Clinic, Graduate School of Medicine, Graduate Hospital, University of Pennsylvania, Philadelphia.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, March 2, 1936.

References to the literature are imperative, for no one person, thanks to what seems to be an ever decreasing incidence of such intracranial complications, has ever seen all of the various possible complications.

The ocular complications seen should appear in the extra- and intraocular muscles, including nystagmus, the oculomotor paralyses and disturbances of the conjugate and associated ocular movements; in the fields of vision and in central visual acuity; in the ocular fundi, especially in the condition of the optic nerve papillae and of the retinal vessels; in certain changes in the extraocular anatomy, as the position of the eyeball in the orbit, pain upon digital pressure to the eyeball, edema of the lids, and lagophthalmos from central types of facial paralysis; and, last, in various less common conditions as visual aphasia and hemiplegia accompanied by homolateral homonymous defects in the fields of vision.

In considering the source or origin of the infection, the first task is a brief review of a part which the sinuses may play in the development of these complications. The maxillary sinus, by reason of its remote distance from the orbit and from the cranial cavity, is the least at fault. Still, Bönninghaus,[1] in a critical research on the intracranial complications of sinus pathology, found in a series of 52 such cases (one cerebral abscess, 27 cases of meningitis and 24 cases of cavernous sinus thrombosis), from two to nine cases which originated either from the maxillary sinus wholly or in which the maxillary sinus played a large part. The frontal sinus, in spite of its contiguity to the brain, is also only rarely to blame, though rather more so than is the maxillary sinus. Cranial osteomyelitis, and extension into the meninges and to the brain substance through this, is naturally excluded. Complications, otherwise, are most uncommon from perforation of the bony walls, but occur usually by means of the venae perforantis. Subdural and extradural abscesses, leptomeningitis, thrombophlebitis and abscesses of the cortex of the brain substance have all been described. The sphenoid sinus is the next most important of the sinuses in etiological importance. Yerger's[2] analysis of this sinus in its intracranial relationship is rather complete. In the years 1911-1920 there were 393 cases of suppurative sinusitis treated at Cook County Hospital. Yerger found 11 cases, or 2.8 per cent, of these to be sphenoidal, and of these 11 cases, seven developed intracranial complica-

tions. Yerger stated that, "while the sphenoidal sinus was the least frequently diseased of the nasal sinuses, it gave rise to the greatest proportion of intracranial complications." In this he must differ with many other rhinologists, though the sphenoidal sinus is rather likely the most common cause of cavernous sinus thrombosis. Of the seven cases, the correct diagnosis of suppurative sinusitis was made in only one, at operation; and in five others, at autopsy; six of the cases had diffuse leptomeningitis, and one case had a temporosphenoidal lobe brain abscess.

This relationship of the sphenoidal sinus to the cavernous sinus is quite important, and in this it is similar to that role which the mastoid and the labyrinth play to the lateral and other immediately continuous and contiguous venous sinuses. The cavernous sinus lies, normally, against the lateral walls of the sphenoid, and may readily become infected, either by direct extension or through one of the perforating veins. In all of these cases of thrombosis of the venous sinuses, it seems that localized vessel wall septic infiltration first occurs, adherent clots develop at these sites of inflammation, continued layers of clot form upon these, one after the other, until the lumen of the vein is closed; and, last, because of the bacterial invasion, necrosis and liquefaction of this coagulum occurs, with the formation of septic emboli, and general systemic pyemia as the end-result. In the similarity mentioned, this thrombotic process of otitic origin may advance to a most extensive degree. Rivière and Bertoin[3] reported a case of mastoiditis in a woman, age 32 years, followed by abscess formation, severe pain, fever and hebetude, notwithstanding operation. The course of the infection continued for over four months, the patient dying in coma. At autopsy, obliterative thrombosis of nearly all the venous sinuses of the dura by organized coagulum was present, the internal jugulars being closed as far as the brachiocephalic trunks.

The ethmoidal sinus is considered as being most commonly at fault in the causation of the various intracranial complications. Apparently the infectious process perforates the lamina cribrosa, and enters the subdural space in this manner. In contradistinction to the frontal sinus, the meningeal complications seem to occur most frequently with the acute phase of suppurative sinusitis of the ethmoid rather than during the chronic course of such infections. In this it is similar to the sphenoidal sinus.

The various oculo-orbital signs and symptoms which may appear in themselves prove nothing as to whether or not there is an extension of the infection into the cranial cavity. They are known to appear, however, in those cases of sinusitis which become complicated by intracranial pathology, hence they acquire an important diagnostic value. In general (and this applies to otitic as well as to rhinologic etiology), the certainty of a developing intracranial infection dates from the appearance of vomiting, of impaired memory and attention, emotional instability, disorientation to the development of coma, transient attacks of aphasia and of similar discrete functions, even though the centres for the functions themselves may not be involved; and, last, the appearance of epileptiform convulsions. It is remarkable, however, how long the interval may be in these cases, at times, from the onset of the peripheral infectious focus to the formation of the intracranial complications.

The otitic complications are even more variable and, if possible, more serious. Almost any portion or anatomical part which lies within the cranial cavity may be affected. Cook County Hospital, in the period of 1911 to 1920, inclusive, had over 1,000 cases of suppurative otitis media. Of the total carefully analyzed by Yerger,[4] 116 cases, or 9.2 per cent, showed at least one of three major complications: meningitis, venous sinus thrombosis, or brain (cerebral or cerebellar) abscess. Meningitis was the most frequent complication, being present in 63 cases, or 50 per cent of the total complications. Thrombosis of the sigmoid sinus was next in frequency, with 34 cases, or 30 per cent of the whole. It is interesting that in this diagnosis of thrombosis, blood cultures were not of material assistance. Of brain abscess there were 23 cases, or 20 per cent of the whole. Operation or autopsy was performed in 17 cases of the otitic brain abscess. Of them, 65 per cent were temporosphenoidal, 23 per cent occipital and 12 per cent cerebellar. Only eight cases of manifest labyrinthitis with spontaneous labyrinthine nystagmus were seen. Of the 63 cases of meningitis, autopsies were performed in 29 cases. Meningitis was present in 100 per cent of them; in addition, sinus thrombosis was present in 34 per cent, undiagnosed brain abscess in 13 per cent, undiagnosed cerebellar abscess in another 13 per cent, and undiagnosed extradural abscess in 10 per cent. In 34 per cent of the total cases,

diagnosed as venous sinus thrombosis, autopsies were done in 14 cases. One hundred per cent of them showed thrombosis of the sigmoid sinus. Meningitis was present in 71 per cent; cerebellar abscess, undiagnosed as such, in 21 per cent; undiagnosed cerebral abscess in 7 per cent; thrombosis of the superior and the inferior petrosal sinuses in 14 per cent; and thrombosis of the internal jugular vein in 14 per cent. The tremendous incidence of meningitis, first, and of brain abscess, second, is quite evident. It seems that Barnhill's postulates[5] are rather all-embracing.

1. Foul smelling pus, cholesteatoma and necrotic bone in the middle ear—suspect extension of infection to the brain.

2. A certain history of the above findings, plus headache, stupor, mental apathy, slow respirations, subnormal temperature, choked disc and other ocular symptoms — a probability of abscess is great.

3. If in addition to the above, intracranial pressure is greatly increased, with numerous cells and with bacteria in the spinal fluid—the presence of abscess is about certain.

In discussing the complications, as they appear, regardless of the exact origin of infection, those manifested in the extraocular muscles will first be considered. The extraocular muscles are rarely involved, and then only in connection with extensive degrees of meningitis. Certain ocular signs do appear, from cervical sympathetic system impairment, which may be an aid in the diagnosis of an intracranial otitic complication. They are of minor importance only, but dilation of the pupil has been observed in occipital lobe pathology. (The cortical centre for the dilator reflex is apparently in the occipital lobe.) Further, those sympathetic fibres which lie about the carotid artery, as the carotid plexus of the cervical sympathetic, may be subject to damage from pathology about the tip of the petrous portion of the temporal bone, resulting in similar peripheral findings. Mydriasis, naturally, if present, would indicate central or peripheral irritation and not a lesion with destructive effects. Such an instance would cause miosis from unopposed contraction of the sphincter iridis. Spastic miosis from IIIrd nerve irritation is a finding not uncommon in meningitis. Nystagmus, ptosis of the levator (that is, partial or even complete IIIrd nerve paralysis), abducens paralysis, and the paralysis of conjugate and associated motions are the most important.

Nystagmus, as it appears, is of the jerky form, and is physiologically and pathologically allied to the lateral rotations of conjugate deviation as seen in destructive, suprapontine, homolateral lesions, or in the suprapontine contralateral irritative lesions. Infrapontine lesions, in general, should reverse the slow phase of jerky nystagmus as they also reverse the position of the eyes in conjugate deviation. While nystagmus is identified by the direction of the quick component, the essential component is the slow phase, a phase which implies a failure of the eyes to maintain a position to which they are voluntarily moved. This relationship of conjugate deviation and of nystagmus is due (given a case of cerebellar abscess) to the relative weakness of deviation to the affected side, so that when the gaze is directed straight ahead there may be a tendency for the eyes to look away from the lesion, owing to the unopposed action of the muscles of deviation to the affected side. When the eyes are directed towards the affected side there is a tendency for them to pull toward the midline. Hence the quick phase of the nystagmus is toward the affected side and the slow phase toward the normal side.

The origin of this slow phase or component must be postulated upon several factors. One is the relationship which it has to the cortical, cerebral and cerebellar control of conjugate movements. Conjugate deviation impulses are conducted from the oculomotor nuclei in the posterior longitudinal bundle on that same side to which the eye may be deviating. A second is the relationship it has to the various oculomotor nuclei, the cortical centres for associated movements, the complex arrangement of internuclear pathways, the substantia reticularis (that dumping ground for all vague and indeterminate involuntary acts), Deiter's nucleus in the medulla, and certain nuclei in the cerebellum, which, together, form the group of vestibular nuclei. Another is the certainty that central or nonlabyrinthine nystagmus shares in the control from these nuclei and pathways, if not wholly with the labyrinthine form when considering pathology, at least in a large part.

The quick phase or component is the natural pull back to a midline position. Sommer and Speigel[6] feel that a centre for this lies also in the substantia reticularis but contralaterally from that side controlling the slow or conjugate deviation phase. They believe, that in addition, the intact labyrinth plays an important part also. Added to this cerebellar pre-

dominance for nystagmus control, as well as conjugate deviation development, the cerebral hemispheres have similar relationships, of a suprapontine or uncrossed fibre type. A laterogyric action of the head and the eyes may be brought about, according to Meyers,[7] by the stimulation of one of two regions in each hemisphere; the first, in the middle and inferior convolutions of the frontal lobe; and the second, in the angular gyrus of the parietal lobe. The abolition of this function from one hemisphere allows, it may be assumed, the laterogyric action of the other to come into play unantagonized and unhindered, producing in this manner a rotation of the head and eyes toward the side of the lesion. Thus, a destructive lesion of the right cerebral hemisphere may be suspected if the quick component of spontaneous nystagmus to the left is absent, *i. e.*, conjugate deviation to the right, or if a conjugate deviation occurs to the right upon labyrinthine stimulation.

In view of the possibility of labyrinthine nystagmus being present in all of these cases, a differentiation of this from central is rather important. Vertical nystagmus, if present, spontaneously, is rather certain to be central and from an abscess. Labyrinthine nystagmus tends to decrease and to disappear as peripheral suppuration continues, while in the cerebellar form it becomes more apparent and stronger as a cerebellar abscess progresses (though herein it also may occur early and then disappear). The cerebellum is probably involved and on the homolateral side when there is a total lack of outward and inward pointing by turning and touching. Spontaneous nystagmus lasting for days with general symptoms as vertigo, staggering gait, asynergia, etc., is more than suggestive of cerebellar involvement. In this cerebellar control of nystagmus each side of the cerebellum is to a certain extent concerned with all positions of the eye, but this control is greater in the direction of the same side and less to the opposite side.

Relative to this, Bing[8] states, in speaking of cerebellar control of oculomotor responses, "in early stages of cerebellar tumors and hemorrhages, etc., there may be, on the contrary, occasionally observed a conjugate deviation to the diseased side. In such instances we are dealing with an irritative symptom." This may be produced experimentally; Bárány's test applied to a lightly anesthetized patient produces a conjugate deviation to the irritated side instead of nystagmus. The abolition of consciousness seems to inhibit the quick component

or automatic return of the eyeball toward its normal position, a movement which is essentially a part of the nystagmus cycle."

Schmeigelow[9] discussed in rather great detail the application of nystagmus to the diagnosis of cerebellar abscess. In his discussion of 14 such verified cases, the correct preoperative diagnosis was made in only five instances. Four other cases were diagnosed as such at autopsy, while in one probable case death occurred without diagnosis or autopsy.

The signs and symptoms not connected with the ophthalmic aspect of these cases are deliberately omitted herewith, except for one bearing an important and serious aspect to these cases. Of the general systemic symptoms, the most frequent of them all — headaches — may be completely absent at times. The other symptoms, as stiffness of the neck, tenderness of the neck on pressure, sensibility of the cranial bones, asynergia and ataxia, etc., these symptoms, while useful in the diagnosis of cerebellar abscess of otitic origin, may be rendered of no importance when the abscess is complicated by meningitis, by osteitis of the occipital bone, purulent thrombosis of the sinuses and suppurative infiltration of the sternocleidomastoid or other muscles of the neck. Vomiting may likewise be absent; on the other hand, it may be present in a great number of conditions having nothing to do with brain abscess. Changes in the ocular fundi, if present are most important, but a large number of cases of cerebellar abscess show no papilloedema, at least for a long time early in the case. Nystagmus seems to be the greatest single factor for diagnosis, according to Schmeigelow. He is quoted herewith verbatim (translation): "It is more important to examine the movement of nystagmus because cerebellar abscess is almost always accompanied by spontaneous nystagmus, more or less accentuated. The latter loses some of its value as a sign in cases where labyrinthine suppuration cannot be excluded. It is generally possible, however, to distinguish cerebellar nystagmus from the labyrinthine type. There are three possibilities: *1.* If there is a nystagmus towards the diseased side, it may be due to circumscribed labyrinthitis, or to cerebellar abscess, but if after operation upon the labyrinth the nystagmus persists toward the diseased side, the cause should be sought in a cerebellar affection. *2.* If a rotary nystagmus is directed towards the well side and the labyrinth is without reaction,

the nystagmus may be due to either the labyrinth or to the cerebellum. Here diagnosis is impossible before operation upon the labyrinth. *3*. Then, if it is due to the labyrinth, it should disappear after two or three days, but if due to cerebellar disease it will remain unchanged, or it may change to the opposite or diseased side, in which case its cerebellar character may be diagnosed with certainty."

MacCuen Smith,[10] in discussing the same problem, shows an even greater diagnostic value for nystagmus; in that it will assist in differentiating labyrinthine from cerebellar, and these, in turn, from that due to cerebral or temporosphenoidal abscess. Quoting him: "Given a case of aural suppuration . . operation or not . . the findings of a spontaneous nystagmus or of a loss of responses from the vertical canals without such a loss from the horizontal canal, or of a perverted nystagmus after douching, at once leads one to suspect strongly some intracranial lesion. When the clinical picture and laboratory tests exclude everything except brain abscess, the vestibular examination might indicate whether such an abscess is in the cerebellum or in the temporal lobe. The ability to elicit vertigo and past-pointing after stimulation will eliminate the cerebellum and suggest the temporosphenoidal lobe as the possible site of trouble." Otologically, and with vestibular stimulation, the absence of a response from the vertical canals, with the presence of a normal response from the horizontal canals, has been a frequent observation in brain stem pathology. Several observers have seen the opposite as well, but this, apparently, is most uncommon. Further, repeatedly, physiologists have obtained a nystagmus response from labyrinthine stimulation after the total extirpation of the cerebellar hemispheres. Hence, it seems certain that the cerebrum, as well as the cerebellum, must be considered as essential to nystagmus, in general; and this at least until the vestibulo-ocular tracts of the slow phase of nystagmus are more definitely delineated. Even if one should agree that the quick phase of nystagmus may be controlled by a lower automatic mechanism, free from cerebral control, the question of the slow phase as applied to man remains unanswered.

Nystagmus and paralysis of conjugate motions are most likely to appear in abscess formation, but the less common paralyses which one sees, of the other associated motions, as convergence and divergence paralyses, and deviations from

horizontal parallelism are likely due to a more or less diffuse meningitis. Convergence and divergence paralyses have been seen, however, with cerebellar abscess. An isolated paralysis of the IVth nerve is considered to be pathognomonic of cerebellar abscess and probably, not through cerebellar involvement so much as by pressure through the quadrigeminal plate. If so, this might also account for some of the rare paralyses seen of associated movements. Eagleton[11] reported a case of temporosphenoidal abscess combined with cerebellar abscess, wherein choked disk, IVth nerve paralysis and aphasia developed with a spontaneous horizontal nonlabyrinthine nystagmus which increased when looking to the left.

The IIIrd nerve, excluding cavernous sinus thrombosis, is the least commonly involved. The IIIrd nerve, after it emerges from the brain stem and before it enters the cavernous sinus, lies free in and passes freely through, the pia and arachnoid spaces. It can be paralyzed, therefore, only when attacked by massive exudate, as one would find in extensive meningitis. Suppuration in the brain stem, sufficient to involve its nucleus, would show other massive changes so gross in symptomatology that localization should not be difficult. Further, the author (and reader) are willing to prophesy that in such cases, an almost inevitable autopsy would confirm this diagnosis. Stout's[12] case of postinfluenzal frontal sinus involvement, accompanied by meningoencephalitis, with iritis, papillitis, and with IIIrd, Vth and VIth nerve involvement, is rather typical of the meningitic process. Linck's[13] case was almost exactly similar, also one of postinfluenzal frontal sinusitis followed by meningoencephalitis. Of the cranial nerves which have been reported by various authors, as involved in the complications of otitis media (the opticus, the oculomotorius, trochlearis, trigeminus, abducens, facialis, acusticus, glossopharyngeus, vagues, accessories and hypoglossus), the IIIrd nerve is the least commonly involved of the oculomotor nerves in abscess, while in neoplasms it is much more frequently involved, due to the mechanics of the intracranial pressure. When it does show paralysis, it has about the same relationship to the probability of temporosphenoidal abscess as has an isolated trochlear paralysis to cerebellar abscess. This was shown very well in a case presented by Adam.[14] In this, nine days after mastoid operation, the temperature rose to 103°, then fell to 97°, followed by a total paralysis of the IIIrd on the

left, with a right-sided facial paralysis, and with aphasia. Reoperation showed the presence of a temporosphenoidal abscess. The aphasia disappeared within a week, as did all other symptoms except that of the IIIrd nerve involvement, and last this also disappeared with ultimately a complete recovery. Adam felt that there was, in this case, a slowly developing encephalitis, rather than an encapsulated abscess. It confirms Macewen's dictum that in cases of crossed paralysis the lesion is subcortical when the contralateral paralysis affects the face more, and ealier than the arm, and arm more than the leg. When the lesion is close to the pons the contralateral paralysis is comparatively uniform and simultaneous.

Abducens or VIth nerve paralysis is perhaps one of the most interesting of all the possible motor paralyses. Eagleton[15] calls special attention to the anatomical conditions which are accountable. It presents the following points of susceptibility: "*1*. As it passes through the bulbar cistern it may be attacked by exudate. *2*. As it enters the dural canal it may be bitten by the sharp dural opening. *3*. In the dural canal a swelling of the periosteum of the apex of the petrous pyramid may paralyze it. *4*. An inflammation of the petrosphenoidal ligament may also paralyze it as it passes through Donello's canal. *5*. It is frequently attacked in the walls of the cavernous sinus itself."

The first two of these dicta are seen not infrequently in cerebellar pathology. The third and fourth postulates are even more common, especially the third, in that it exists as one of the triad which constitutes the Gradenigo[16] syndrome. This syndrome, reviewed in detail by Sears,[17] is well known and not real uncommon. If it is truly present, spontaneous recovery is rather certain, nor does it have much prognostic value. Its appearance after surgery does not demand further surgery, except with the development of other signs and symptoms pointing to subdural abscess or to a frank osteomyelitis of the petrous portion of the temporal bone. Under such circumstances it is no longer a simple Gradenigo syndrome, but now a part of the symptomatology of pyramid osteomyelitis or some similar and equally grave complication. Its development prior to any surgery has a different prognostic value or aspect.

Lapouge[18] who did much work on this syndrome, is being quoted herewith (translation), in that the author feels that

at least from an ophthalmological standpoint, he holds a very logical attitude toward this syndrome. "There probably exists two different forms of the Gradenigo syndrome, one benign and frequent, and one malignant, less common, and almost always fatal. Two different petrous and meningeal lesions must correspond to these two different forms. A simple cellulitis of the petrous bone and a simple meningeal congestion are probably responsible for the former. On the other hand, the presence of an osteitis of the anterosuperior petrous surface, and at the tip an extradural abscess or intermeningeal abscess, probably, constitute the lesion of the malignant variety. The first requires free drainage of the tympanic cavity; the second, intervention at the petrous bone. In the course of a Gradenigo syndrome, and after free drainage, if drainage persists, if the paralysis of the VIth remains complete, if the pulse is unstable and the patient generally unimproved, and especially if there is a fistula of the attic or promontory, extradural abscess complicating the lesion should be suspected."

Relative to the Vth nerve facial pain, which accompanies it, as part of the syndrome, Eagleton[15] has written in great detail. He called attention to the longer course which the ophthalmic branch has, its firm adherence to the dura and to the cavernous sinus, and to the fact that its ganglionic portion lies flat on the dura adherent to the bone of the petrous apex, while the ganglionic portions of the second and third divisions of the Vth nerve lie on the elastic cushion formed by the internal carotid artery as it runs in the open foramen of the carotid canal. Vail[19] felt that this pain was not due to irritation of the Vth, but due to irritation of the great superficial petrosal nerve. Regardless of these differences of opinion, this fact remains, that VIth nerve paralysis is one of the most common signs of otitic (and rhinologic) intracranial extension. This facial pain is perhaps closely related to the old statement, "that pain behind the eye, if and when it appears during the course of otitis media, is indicative of intracranial extension of infection into the middle fossa." Further, it is the basis (and we assume it to be from Vth nerve superior branch irritation) upon which Eagleton[15] made the statement, "consequently, on anatomic grounds, and from clinical experience, pain behind the eye may be the first manifestation of congestion or granulation caries of the

petrous apex." Johnston's[20] recent detailed review of the condition is well worth reading. He considered 45 additional cases, showing 17 per cent (eight cases) of deaths, this being about the same rate as found by Sears in his earlier review of 250 such cases.

The fifth of Eagleton's postulates refers to cavernous sinus thrombosis. With this discussion we will consider the other related intracranial venous sinuses. Burger[21] reviewed 49 cases of otogenic lateral sinus involvement and 61 cases of rhinogenic thrombosis. Of the 49 otogenic cases, 25 recovered, while of the rhinogenic cases none recovered. (Recoveries have been obtained, however, in the author's experience as well as in that of various writers.) Burger felt that the starting point of lateral sinus thrombosis was always the inner and middle ear, while in cavernous sinus involvement, the sphenoid was most commonly at fault, and that phlebitis of the superior longitudinal sinus started almost exclusively with the frontal sinuses. Ophthalmologically, the cavernous sinus involvement shows the greatest amount of symptomatology. The IIIrd, IVth and VIth nerves show an early and complete paralysis, proptosis of the eyeball develops and chemosis of the conjunctiva appears early, and advances rapidly, and to a great degree. The fundi show rather commonly an engorgement of the retinal veins and inflammatory edema of the papilla, even with hemorrhages; and while the condition very rarely may remain unilateral, the usual bilateral ocular involvement, within 24 to 48 hours, shows the rapid extension of the thrombosis throughout both sides of the cavernous sinus. The lateral sinus cases, occasionally, show similar ocular fundus changes, and occasionally unilateral, and less rarely homonymous, cuts in the contralateral fields of vision; still these findings have in no way the absolutely specific localization value possessed by those just mentioned for the cavernous sinus. Occasionally an embolus of high virulence may leave the lateral sinus and become impacted in the middle cerebral artery, leading to the rapid development of cerebral abscess, the abrupt development of contralateral hemiplegia, and in these instances to nearly complete contralateral homonymous field defects.

The fields of vision, if carefully and frequently studied, can be of great importance in certain of these various complications. It is a characteristic feature of them, in such intra-

cranial complications, that they change rather often. Further, field changes will many times antedate other signs and symptoms of a much more serious import. Temporosphenoidal lobe and lateral sinus thrombosis are more likely to have field changes in the highest percentage of cases, and when present, the defects are commonly homonymous and in the contralateral fields of vision. Occipital lobe abscess, while of themselves the most rare, should show this in the highest percentage of cases. Cerebellar abscess may show field changes, but if so, will present nothing of great localizing value, usually a simple concentric contraction. The presence of field changes is most valuable in diagnosis; their absence, however, is not to be taken as an indication of normal intracranial conditions. This is equally important.

The ocular fundi and, more specifically, the optic nerve papillae, have an exactly similar relationship to intracranial pathology. Approximately 50 per cent of cases with subtentorial complications will show a choked disc from the intracranial pressure. Generalized edema of the brain is much greater with brain abscess than with neoplasm, hence papilloedema should be seen fairly frequently. Lateral sinus thrombosis and temporosphenoidal lobe pathology are next most frequent in presenting this definite sign, while abscess of the frontal lobe shows it most uncommonly. Optic nerve neuritis, and not papilloedema, is seen perhaps more commonly in cases of generalized meningitis. From autopsy findings, however, meningitis is itself so often complicated by undiagnosed abscess, that this may not be of any great diagnostic importance. To recapitulate here as above, positive optic nerve papillary changes, when present are a valuable sign, but if absent are not a guarantee of normalcy. So many factors enter into the formation of papilloedema. The amount and locality of the intracranial edema and pressure, the rate or rapidity with which this develops, the virulence of the infection, the degree of intracranial vascular disturbance, the intraocular tension and the susceptibility of the optic nerve to toxins and to bacterial invasion—these all are indeterminable factors, and each of them plays an individual and indeterminable role. As both papillitis and papilloedema have been found, the above statement is conclusively proven.

Frontal lobe abscess seems to present most commonly local pathological changes in the lids and the orbita. Lingaard[22]

reviewed four cases of abscess of the frontal lobe. They all showed such changes, including orbital phlegmon, abscess of the lid, edema of the lids and the conjunctiva, exophthalmos, and in the fundus, vascular changes which one might expect with the disturbances in peripheral venous circulation. If this is so, in the experience of others, it is valuable, for frontal lobe abscess is notably poor in the presentation of any specific localizing signs of symptoms.

Motor aphasia has been mentioned in its relationship to left-sided temporosphenoidal abscess. It is not common; also it has been reported as being present, as well, with lateral sinus thrombosis. Eagleton stated that generalized edema of the brain, without involvement of the speech center, might also be responsible for its occasional appearance.

In conclusion, five cases from the literature are very briefly abstracted and compared to illustrate the value of the possible ophthalmological findings. The first two were, we agree, undiagnosable and came to autopsy, the last three were diagnosed and operated upon with recovery in each instance.

(Hadfield[23]): A child, age 8 years, when admitted was mentally confused, had continual headaches and photophobia, rigidity of the posterior cervical muscles, and with great tenderness. Both tympanic membranes were perforated. There was an optic nerve swelling of 1.5 diopters in each optic nerve disc. Spinal fluid showed moderately increased pressure, with 12 cells per cm., 70 per cent being polys. The postmortem examination showed an abscess, the size of a pea, containing thick yellow pus, in the substance of the optic thalamus. Both middle ears also contained pus. The second, a boy, age 12 years, was admitted, complaining of headaches and of vomiting; shortly after admission and eight days before death he went into coma, dying during a series of epileptiform convulsions. Both optic nerve papillae showed a marked papilloedema. The spinal fluid showed a considerable increase in pressure, and had 12 cells per cm., with 75 per cent of them polys. The autopsy showed a large unruptured abscess to be present in the right frontal lobe. No other signs or symptoms appeared. In the third case (Hautant, Ramadier and Lanos[24]) there was an enormous temporal lobe abscess without any signs of localization, no aphasia of any type, though the site of the abscess was on the left, neither somnolence nor oph-

thalmoscopic signs, and at operation no signs nor macroscopic evidences of osteitis. Lack of co-ordination of temperature and of the pulse were the only definite signs. The fourth case presented an abscess in a similar location, with headache, vomiting, sleeplessness, incoordination of temperature and pulse, paresis of the extremities upon the opposite side, and with a high degree of choked disc. No. 5 was a case of an abscess of the cerebellum with a very complex symptomatology, incuding choked disc, paralysis of convergence and of divergence, facial paralysis, and with nystagmus.

In comparison, neither the absence nor the presence of ophthalmological signs seemed to be characteristic of or classical to these five cases quoted. Two cases with positive eye findings recovered, and a fifth (Case 3), with barely any localizing signs or symptoms, also made an uneventful recovery. The moral is almost too evident to need vocal expression. The author feels that ophthalmologists and otorhinologists will do well to work together, in the greatest possible cooperation, to keep down the mortality rate in these cases of rhinologic and otitic intracranial complications. These patients are handicapped before the race has even begun and need every possible assistance which we together can give.

BIBLIOGRAPHY.

1. Bonninghaus: *Handbuch der Spec. Chir.*, 2nd. Ed., 1914, Katz, Preysing, Blumenfeld.

2. Yerger: *Ill. M. J.*, 43:304, April, 1923.

3. Reviere and Bertoin: *Lyon Chir.*, 19:279, Nov.-Dec., 1922.

4. Yerger: *Eye, Ear, Nose and Throat Monthly*, 3:385, Aug., 1924.

5. Barnhill: *Nose, Throat and Ear*, Appleton, 1928, p. 551.

6. Speigel and Sommer: *Ophthalmo- and Oto-neurol.*, Springer, Berlin, 1931.

7. Meyers: *Amer. J. of Med. Sci.*, 5:169, May, 1925, p. 742.

8. Bing: Compendium of Regional Diagnosis, C. V. Mosby, 1927, 3rd Ed., p. 147.

9. Schmiegelow: *Bull. d'oto.-rhino.-laryngol.*, etc., Paris, 21:49, March, 1923.

10. MacCuen, Smith: The Laryngoscope, 34:534, July, 1924.

11. Eagleton: *Arch. Otolaryngol.*, 10, Dec., 1929, p. 650.

12. Stout: *Penn. Med. J.*, Jan., 1935 (Influenzal Frontal Sinusitis).

13. LINCK: *Ztschr. f. Hals., Nasen. u. Ohrenheilk.*, Berlin, 7-129, No. 2, 1924.

14. ADAM: *Brit. Med. J.*, London, July 24, 1922, p. 991.

15. EAGLETON: *Arch. Surg.*, 20, March, 1930, pp. 386-420.

16. GRADENIGO: *Arch. f. Ohrenheilk.*, 62:255, 1904.

17. SEARS: *Tr. Amer. Acad. Ophthal. and Otolaryngol.*, 29:264, 1924.

18. LAPOUGE: *Rev. de Laryngol., etc.*, Bordeaux,44: 763, Sept. 30, 1923.

19. VAIL: THE LARYNGOSCOPE, 32:569, Aug., 1922.

20. JOHNSTON: *Ann. Otol., Rhinol. and Laryngol.*, 43:1, March, 1934.

21. BURGER: *Acta, Otolaryngol.*, Stockholm, 6:4, No. 1-2, 1924.

22. LINDGAARD: *Ann. Otol,, Rhinol. and Laryngol.*, 28, 1919, p. 129.

23. HADFIELD: *Lancet*, London, 205:929, Oct. 27, 1923.

24. HAUTANT, RAMADIER AND LANOS: *Ann. d. mal. de l'oreille, due larynx*, etc., Paris, 43:1074-1083, Nov., 1924.

1930 Chestnut Street.

NEUROLOGICAL ASPECTS OF FRONTAL LOBE ABSCESS.*

DR. CHARLES E. CONNOR, St. Paul, Minn.

The occurrence, in my practice, of two cases of abscess of the frontal lobe, one entirely without symptoms, though its presence was suspected and every effort made to establish its diagnosis before operation, roused my interest and led to an attempt to see how true was Gerber's statement (quoted by Leegaard[12]) that "the common symptom of frontal lobe abscess is its absence of symptoms." A search was made, therefore, through the records of the 25 local hospitals, and 20 cases of frontal lobe abscess which had never been reported in the literature were found; most of these had been discovered at operation on the frontal sinus, in exploratory proceedings for undiagnosed intracranial lesions, or at autopsy.

The records of the pathological department of the State University, covering a period of 35 years and 22,700 autopsies, were next checked; the reports found, however, had already been obtained from the hospital records examined.

The next step was a search of all available English and American literature for reports which might be those of frontal lobe abscess. Only cases were considered in which the presence of a solitary abscess had been definitely established, either by operation or postmortem; many cases of multiple abscesses were rejected because of the element of confusion introduced into the neurological picture by such lesions. Two hundred four cases fulfilling the above requirement were found.

These case reports were critically reviewed for neurological signs; the findings constitute this paper and will be discussed under the following headings:

A. General discussion. *B*. Cranial nerves to extrinsic ocular muscles. *C*. Cranial nerves and sympathetic system to

*Presented as Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, 1935.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, March 14, 1936.

intrinsic ocular muscles. *D.* Optic nerve. *E.* Pyramidal tract. *F.* Motor cranial nerves not elsewhere discussed. *G.* Sensory disturbances. *H.* Reflexes. *I.* Aphasia. *J.* Psychic changes.

A. GENERAL DISCUSSION.

The diagnosis of frontal lobe abscess has always been attended with such difficulty and so many cases have been recognized only at the postmortem table that the tradition has come down in the literature that frontal lobe abscess is diagnosed by its absence of symptoms. Leegaard[12] remarks that "in these cases (frontal lobe abscess) as a rule no focal symptoms are to be found; in general, in making the diagnosis, we must be content with the etiology of the case (the sinusitis), combined with the more or less distinct signs of intracranial pressure. In this way diagnosis has in many cases been successfully made; but this nearly always demands a certain time for observation, and often the mode of procedure in the first place is to operate for the sinusitis. If the symptoms do not disappear, as had been expected, if they continue and increase, it gradually becomes clear that there exists something more than an inflammation in the frontal sinus, and then finally the cerebral abscess is diagnosed. Such a state of affairs is so typical that on reading through the history of cases we find these conditions recurring again and again, almost with the regularity of a law." Skillern[21] bore witness to this truth when he said, "I do not recall a case where the operator opened up the frontal abscess and drained an abscess—I mean a blind brain abscess in the frontal lobes —that was diagnosed primarily."

Absence of symptoms is more characteristic of the right than of the left frontal lobe, owing to the presence of speech centres in the latter; Neumann[17] recognizes this difference when he observes, in speaking of otitic brain abscess, that "the prognosis for right-sided abscesses is better, since we have not to wait for the aphasia, the characteristic symptom of left-sided abscess. On the contrary, we are obliged to make our diagnosis on the basis of general symptoms, and therefore make an exploratory brain puncture much earlier than in cases of left-sided abscess with aphasia." If this be true of otitic temporal lobe abscess it should hold to a certain extent for abscess of the frontal lobes.

Even as Neumann stresses the importance of general signs of brain abscess, so Macewen[13] called attention to the value of local symptoms: "When symptoms were present pointing to a lesion in one of the recognized cerebral centres, it was better to depend on them as guides, at least to the encephalitis surrounding the abscess, than to trust to the indications afforded by the scalp or skull injury."

The need and value of localizing signs, always great in any intracranial lesion, become doubly so in the presence of bilateral frontal sinusitis; confusion arises from the fact that the sinusitis may produce symptoms closely resembling those of the complication, so that lateralization of the latter becomes difficult or impossible; in such a situation the faintest localizing sign becomes important.

Experience teaches that frontal lobe abscess may attain great size without symptoms; Leegaard[12] observes that "we can with fairly great certainty count upon the cerebral abscess not being far distant from the cerebral wall of the frontal sinus. It is, therefore, directly behind this latter that we must look for the abscess. These abscesses, by the time we are in a position to diagnose them with any approach to certainty, have as a rule attained pretty large dimensions." Halstead and Vaughan[10] thought that these abscesses frequently occur low in the second and third frontal gyrus and do not, therefore, involve motor tracts and centres as frequently as do tumors, which are apt to be higher up and further back; abscess has a tendency to be to the front and median line.

Abscess of the frontal lobe produces neurological signs by pressure upon or destruction of, nerve centres and tracts. Destruction of tissues by inflammation is always a local process; whether or not localizing symptoms result depends on the presence of structures (centres and tracts) in the area involved, destruction of which is attended by such symptoms. Increased intracranial pressure, caused by the abscess itself, or by its attending edema, congestion, or encephalitis, may be direct or transmitted, localized or generalized, of slight or of great degree. Direct pressure is exerted upon contiguous structures, is localized, and produces localizing symptoms if important structures are involved, *e. g.*, pressure upon the precentral gyrus with resultant Jacksonian attacks, pressure

upon the olfactory nerve with resultant anosmia. Transmitted pressure may manifest itself by symptoms arising from involvement of structures at a distance, *e. g.*, hemiplegia from an abscess in the frontal pole of the frontal lobe. Direct or transmitted pressure may be of slight degree, causing symptoms of irritation, such as twitching, spasm, motor weakness (motor nerves), or paresthesia (sensory nerves); or of great degree, causing convulsions, paralysis or anesthesia.

Increased size of the frontal lobe, evidenced by tense dura and flattened gyri, may cause mass displacement of the cerebrum with its resultant compression against the cranial walls, especially the floor; the medulla may be compressed as it passes through the tentorium.

Absence of symptoms may be attributed, not only to the fact that important centres and tracts are not involved, but also to the fact that the flow of cerebrospinal fluid is not obstructed; absence of firm dural attachments, while permitting displacement of the cerebrum, may also prevent undue stress from such displacement.

B. EXTRINSIC OCULAR MUSCLES.

Paresis or paralysis, much less commonly spasm, of the extrinsic ocular muscles is seen in increased intracranial pressure and hence in large frontal lobe abscess; the opinion is quite generally expressed that such paralysis has little localizing significance and indicates increased pressure only. Eagleton[6] makes the statement that oculomotor paralysis has little, if any, localizing value, that trochlear paralysis localizes the lesion in the posterior fossa near the midline, and that abducens paralysis stands between the two in localizing importance. Fuchs[8] states that paralysis of the individual trochlear or abducens nerve has little or no localizing value, that involvement of the oculomotor nerve may permit of some localization by studying the distribution of the lesion among its branches; Coleman[5] feels that ocular palsies are of little importance.

Involvement of the extrinsic ocular muscles in increased intracranial pressure is easily understood when one considers the anatomy of their nerve supply. The nuclei of the oculomotor and trochlear nerves are in close proximity in the mid-

brain beneath the corpora quadrigemina and the aqueduct of Sylvius; the abducens nucleus is a centimeter lower down in the pons, close to the genu of the facial nerve under the floor of the fourth ventricle. This entire area is vulnerable to increased pressure. The nerves themselves are frequently involved in their intracerebral courses from their nuclei, and in their intracranial courses from their points of superficial origin in the brain stem to their exits from the skull through the superior orbital fissure. The exits from the brain of the oculomotor and abducens nerves are near the pyramidal system; the abducens may actually pass through this tract and the trochlear nerve curves around it. These relations explain the frequent association of motor disturbances of the extrinsic ocular muscles with those of the contralateral side of the body.

The cranial nerves, through their nuclei and the pyramidal tracts, have bilateral representation in the motor cortex, mostly contralateral, as a result of which a unilateral lesion in the upper motor neuron does not usually cause paralysis, a wise provision on the part of Nature to protect such vital functions as swallowing and breathing. It is not necessary, therefore, to consider the supranuclear tracts and the motor cortex in attempting an explanation of the ocular palsies occurring in frontal lobe abscess; they must be peripheral, in the intracerebral or intracranial portion of the nerve.

These nerves may be divided into zones to facilitate discussion of the localizing value of their lesions. Whitnall[24] suggests that any orderly treatment consider successively the nucleus of origin, the intracerebral course, the superficial origin, the intracranial course, the path through the cavernous sinus, the orbital course, and the final distribution (muscles). Fuchs[8] divides paralyses into cerebral (brain), peripheral (peripheral nerves), and muscular (muscles), or intracranial and orbital. Finer division of the intracranial group would include cortical, supranuclear, nuclear and fasicular (nerve trunk) lesions.

I. OCULOMOTOR NERVE.

The oculomotor nucleus consists of several paired and one unpaired group of ganglia. The former constitute the large-celled lateral chief nucleus of Whitnall, supplying the extrinsic muscles of the globe, and are subdivided from before back-

ward into smaller groups of cells supplying individual muscles, levator palpebrae superioris, rectus superior, rectus inferior and obliquus inferior; the unpaired ganglia, composed of two smaller-celled nuclei in front (Edinger-Westphal nucleus) and one behind and below, fused with its fellow of the opposite side to form a central mass, supply most of the intrinsic muscles of the eye (sphincter of the iris and the ciliary muscle).

It is generally agreed that fibres destined for the levator palpebrae superioris and rectus superior are entirely uncrossed, and those to the rectus inferior entirely crossed. Disagreement exists, however, as to the composition of the nerves to the rectus internus and obliquus inferior. Fuchs[8] represents the fibres of the rectus internus as entirely uncrossed, while Whitnall[24] considers them to be both crossed and uncrossed. Fuchs represents the fibres of the obliquus inferior as entirely crossed, while Whitnall considers them to be both crossed and uncrossed. This division of the oculomotor nucleus and disposition of its fibres has not been certainly demonstrated in man but is thought to hold for apes.

From its superficial origin in the oculomotor sulcus above the pons the nerve runs a course of 25 mm. in the posterior fossa to enter the cavernous sinus, through which it courses to pass into the orbit through the superior orbital fissure. Paralysis may result from injury at any point in its entire course.

Table 1 summarizes the lesions of the oculomotor nerve.

The terms ipsilateral and contralateral, as used throughout this study, refer to the side of the cerebral abscess.

A lesion involving the intracranial portion of the oculomotor nerve might logically be expected to involve other nerves in the immediate vicinity, such as the trochlear; in three cases in which such a lesion was probable (two of complete external ophthalmoplegia and one of complete oculomotor paralysis), there was one example of paralysis of the ipsilateral trochlear nerve. The contralateral facial nerve was involved once, the ipsilateral pyramidal tract twice.

Again, it is difficult to picture the site of any isolated lesion, either in the intracranial course or nucleus of the oculomotor

nerve, which could involve only the fibres destined for an individual muscle. Yet, eliminating from the above group of 22 cases of oculomotor involvement the one case of total oculomotor paralysis, 21 are left in which only partial involvement of this nerve was present; two of these were cases of total external ophthalmoplegia.

TABLE 1—OCULOMOTOR LESIONS.

Nerve	No. of Cases	Other ipsilateral oculomotor lesions				Abducens paralyses		Facial paralyses		Ipsilateral pyramidal tract	Ipsilateral complete ext. ophthalmoplegia	Orbital disease
		Dilated pupil	Superior rectus paralysis	Internal rectus paralysis	Complete oculomotor paralysis	Ipsilateral	Bilateral	Ipsilateral	Contralateral			
Levator palpebrae superioris	16	6			1	2	1		4	8	2	2
Superior rectus	4	1		1				1	1			3
Internal rectus	2	1	1									
Total	*22*	*8*	*1*	*1*	*1*	*2*	*1*	*1*	*5*	*8*	*2*	*5*

SUMMARY OF OCULOMOTOR LESIONS.

Involvement of the oculomotor nerve was present in 22 of 204 cases of frontal lobe abscess. In only one case was the paralysis total. In all others it was partial and manifested most frequently, in the order named, by paralysis of the levator palpebrae superioris, rectus superior and rectus internus.

Other neurological signs associated with lesions of the oculomotor nerve were contralateral motor disturbances of the body, due to involvement of the ipsilateral pyramidal tract above its decussation, dilatation of the ipsilateral pupil, and paresis of the contralateral facial nerve.

Difficulty arises if one attempts to localize the site of the lesion in this group of 22 cases; Fuchs[8] remarks that complete oculomotor paralysis almost certainly involves the intracranial

course of the nerve proximal to the superior orbital fissure; it could scarcely be nuclear, owing to the fact that the internal rectus and inferior oblique muscles are partially, and the inferior rectus muscle wholly, activated by crossed innervation and would, therefore, in an ipsilateral nuclear lesion, receive stimulation from the contralateral normal nucleus.

Consideration of these facts permits only the conclusion that lesions of the oculomotor nerve, alone or associated with involvement of the pyramidal tract or facial nerve, have nc exact localizing value, but point only in a general way to the middle or posterior fossa of the skull as the probable site of the lesion. They have some lateralizing value, in that they occur more frequently on the side of the frontal lobe abscess than on the opposite side.

II. TROCHLEAR NERVE.

The trochlear nerve's intracranial course is long (40 mm.), largely because it emerges from the posterior side of the brain stem, about which it swings to join the oculomotor and abducens nerves; it passes through the cavernous sinus and enters the orbit through the superior orbital fissure to supply the superior oblique muscle. Trochlear paralysis results in inability to depress and intort the eye. The nerve is rarely affected alone; only one such involvement occurred in this study.

Eagleton[6] states that trochlear paralysis localizes the lesion in the posterior fossa near the midline. Its common involvement with other nerves to extrinsic ocular muscles carries with it the localizing and lateralizing values attributed to lesions of those nerves.

III. ABDUCENS NERVE.

There were six cases of ipsilateral abducens paralysis, two of contralateral, and three of bilateral involvement, in 204 cases of frontal lobe abscess.

The statement regarding the lack of localizing value of paralysis of the extrinsic ocular muscles applies with especial force to abducens paralysis. It has a long intracranial course and is especially liable to trauma on account of its exposed

position on the ventral surface of the pons, where there is very little space between the bony wall and the brain. In increased intracranial pressure it is apt to be compressed against the pons by the posterior cerebral branch of the basilar artery, which is pushed down against the inferior olive. Sixth nerve palsies, common in fracture of the base and in petrous tip suppuration, have, therefore, but little localizing value. The fibres are entirely uncrossed.

Table 2 records the lesions of the abducens nerve.

TABLE 2 — ABDUCENS LESIONS.

Side	No. of Cases	Oculomotor paralyses					Facial paralyses		Ipsilateral pyramidal tract	Orbital disease
		Ipsilateral		Contralateral	Bilateral		Ipsilateral	Contralateral		
		Ptosis	Fixed dilated pupil	Fixed contracted pupil	Dilated pupils	Ptosis				
Ipsilateral	6	1[a]						1	1	4
Bilateral	3		3	2		3		3	3	1
Contralateral	2	1			2		2			
Total	*11*	*2*	*3*	*2*	*2*	*3*	*2*	*4*	*4*	*5*

a. Probably orbital due to foreign body abscess

The occurrence of orbital disease in four of six cases of ipsilateral abducens palsy raises the question as to whether the lesion was orbital or intracranial; the presence, in one case, of involvement of the contralateral facial nerve and ipsilateral pyramidal tract points to the middle or posterior fossa, rather than to the orbit, as the site of the lesion. There were no associated signs in the other three cases to indicate definitely whether the lesion was orbital or intracranial.

One case presented an ipsilateral ptosis which was, however, probably peripheral in origin, inasmuch as there had

been a foreign body orbital abscess with considerable lid trauma.

Ipsilateral abducens paralysis occurred twice without orbital disease; the absence of facial nerve involvement in these cases points more to the intracranial portion of the nerve than to its nucleus as the site of involvement because the nucleus of the abducens is in contact with the genu of the facial nerve and its disease is usually accompanied by disturbances in facial musculature. This is not always true, however, because pressure may not equally affect gray matter (nuclei) and white (fibres); its exertion, in nondestructive lesions, on the facial colliculus (prominence on the floor of the fourth ventricle, where the facial nerve turns around the nucleus of the abducens) might cause a lesion of one nerve and not of the other.

SUMMARY OF ABDUCENS LESIONS.

Lesions of the abducens nerve were most frequently associated with involvement of the ipsilateral pyramidal tract, contralateral facial nerve, or oculomotor nerve of either side; unilateral lesions of the oculomotor nerve were most frequent on the ipsilateral side.

All cases presented definite evidence in fundi and reflexes of considerable increase in intracranial pressure and it is only logical to assume that this pressure affected the nerves in question on each side, either directly or indirectly.

The only significance which may, in cases of frontal lobe abscess, be attached with certainty to lesions of the abducens nerve is that of increased pressure in the middle or posterior cranial fossa.

Such lesions have some lateralizing value, in that, when unilateral, they occur more frequently on the side of the cerebral abscess than on the side opposite the abscess.

SUMMARY OF PARALYSES OF EXTRINSIC OCULAR MUSCLES.

Paralyses of extrinsic ocular muscles may be caused by disease of the orbit. Their occurrence, in cases of frontal lobe abscess not so complicated, is due to increased pressure in the middle or posterior fossa of the skull. Their frequent combination with motor disturbances in the field of either

facial nerve or of the pyramidal tract, usually of the same side, also points to the pons or midbrain as the site of the lesion.

The more frequent occurrence of unilateral ocular palsies on the side of the cerebral abscess is of some lateralizing value, which, however, is limited by the occurrence of such lesions on the side opposite the abscess.

Bilateral ocular palsies are indicative only of increased intracranial pressure and have no localizing value.

The nuclei and intracerebral portions of the oculomotor, trochlear and abducens nerves are in such close proximity in the midbrain and pons and their intracranial segments on the base of the skull in such intimate relationship that increased intracranial pressure does not produce a neurological picture of distinctive design into which any exact localizing or lateralizing significance can be read.

The incidence of ocular palsies was not appreciably influenced by the presence of orbital disease.

IV. CONJUGATE DEVIATION.

Conjugate deviation of the eyes with or without associated turning of the head, either to the same or opposite side, is a phenomenon frequently mentioned in connection, not only with frontal lobe abscess, but also with other diseases, such as epilepsy. Halstead and Vaughan[10] observed conjugate deviation of the head and eyes premonitory to Jacksonian epilepsy; Osler[18] and Atkinson[1] noted the same finding. In this series of cases conjugate deviation of the eyes was noted three times, twice to the side of the lesion and once to the opposite side. Conjugate deviation of the eyes and turning of the head to the side of the lesion was noted but once. In none of these three cases did the accompanying neurological findings form any characteristic picture.

Whether or not conjugate deviation of the eyes has any localizing value depends, of course, on its mechanism. This is to be found in the oculomotor, trochlear and abducens nuclei, the posterior longitudinal bundle, and certain special centres which are thought to act as coordinating and distributing points for the impulses concerned in conjugate movements;

the innervation of the recti muscles (the internal rectus of one side and the external rectus of the other), is especially important in lateral movements.

Ballance[3] states that the centre concerned with movements of the head and eyes is located in the area between the posterior end of the second left frontal convolution (containing Charcot's motor graphic area) and the posterior end of the third left frontal convolution (Broca's speech centre); he quotes Mills to the effect that the symptom complex of the region is motor agraphia, motor aphasia, fits, movements of the head and eyes, and psychic changes. Grinker[9] mentions not only this centre, stating that it is probably that for voluntary movements of the head and eyes, but also a centre in the occipital lobe, concerned chiefly with reflex changes in eye positions, and adds that these centres are antagonistic, irritative lesions producing movement toward the opposite side, and destructive lesions, movement toward the same side.

Riley[20] describes special co-ordinating centres for lateral and vertical conjugate eye movements and for convergence; a special centre for divergence has not been demonstrated.

The centre for the control of lateral conjugate movement is thought to be near but not identical with the abducens nucleus, sending fibres directly to the homolateral abducens nucleus and to the contralateral oculomotor nucleus through the posterior longitudinal bundles.

Spiegel,[22] in a recent paper, reports experimental work on cats which suggests strongly that the impulses from the frontal lobe motor centres for eye movements, after passing through the pyramidal tracts, enter the vestibular nuclei. Here they are redistributed to enter the ascending fibre tracts of the posterior longitudinal bundles of each side to reach the abducens and oculomotor nuclei. Riley[20] also mentions the role of the vestibular nuclei in lateral deviation, stating that impulses pass, chiefly from the nucleus of Deiters, directly across the tegmentum to enter the posterior longitudinal bundle of each side and end in the special centres controlling lateral deviation.

The superior colliculus, according to this author, may be considered the special centre or redistribution point for impulses controlling vertical deviations; the mesial portion

of that segment of the oculomotor nucleus innervating the internal rectus muscle, the nucleus of Perlia, is considered to be the special centre for convergence.

In speaking of the importance of the posterior longitudinal bundle in conjugate movements, Riley says: "Its constituents number, not only the internuncial fibres passing between the various centres already mentioned and between the various nuclei of the oculomotor apparatus, but also the fibres that are concerned with the reflex activities of the eye movements, these being chiefly represented by the trigeminal, the acoustic, and the vestibular contributions. It also serves to transmit the impulses that are received from suprasegmental sources that control the associated, synergic and voluntary control over the oculomotor movements."

Meyers,[15] in a recent paper, finds conjugate deviation of the eyes a valuable diagnostic sign, especially in unconscious patients or in cases in which no history can be elicited, and observes that it is less frequently noted in tumor than in abscess because the slow development of the tumor permits compensatory action of other centres which corrects the deviation; shifting and stretching of the entire brain also minimizes the effect of tumor. In a rapidly developing abscess the persistence of conjugate deviation in increasing intensity is of value and should always be looked for.

Meyers' conclusions are of value: "In abscess of the cerebrum conjugate deviation of the head and eyes is extreme when the lesion affects the angular gyrus of the parietal lobe; it is of moderate extent if the lesion affects the posterior part of the frontal lobe close to the Rolandic area. It is absent, no matter how extensive the lesion, in abscess of the prefrontal region of the frontal lobe, in abscess of the occipital lobe, and in abscess of the temporal lobe. The deviation in cerebral lesions, when present, is toward the side of the lesion. Interpreting the movement as an attitude of attention and assuming the angular gyrus to be the centre of psychic vision (*e. g.*, the interpretation of visual stimuli), as shown by the fact that lesions of this region on the left side give rise to alexia or agnosia, the inability to understand writing or to recognize objects by vision, I conclude that attention is dominated most powerfully by stimuli arising within the sphere of vision and relatively little by other kinds of stimuli."

Anyone interested in this subject is referred to Meyers' most excellent article.

Apparent agreement exists that conjugate deviation of the eyes is caused by supranuclear lesions; the fact that the internal rectus involved in conjugate deviation may function normally on convergence also places the lesion above the level of the nucleus.

Conjugate deviation of the eyes, especially when associated with evidence of cortical irritation, may be caused by irritation of the centres concerned and is probably, therefore, toward the side opposite the cerebral lesion. When associated with evidence of severe cortical damage, such as paralysis, it may be caused by paralysis of the centres involved and is probably, therefore, toward the side of the cerebral lesion.

It has value, when taken in conjunction with other findings, not only in lateralizing the lesion but also, in certain cases, in pointing to definite areas within the frontal lobe itself.

C. INTRINSIC OCULAR MUSCLES.

Involvement of the intrinsic musculature of the eye is revealed by the state of the pupil, which is the net result of the interplay of stimuli upon the sphincter and dilator mechanisms of the iris.

I. DILATATION.

Active dilatation of the pupil (spastic mydriasis) is effected through the superior colliculus, the medial longitudinal bundle of the same side, the tectospinal fasiculus of the opposite side, Bridge's ciliospinal centre in the intermediolateral cell column of the eighth cervical and first, second and third upper thoracic segments of the same side, cervical trunk, superior cervical ganglion of the same side and the internal carotid plexus. At this point the fibres divide; most of them go through the caroticotympanic nerve (or plexus), the cavernous and ophthalmic plexus, and both the long and short ciliary nerves to the dilator of the iris. A smaller number proceeds through the semilunar ganglion, the ophthalmic division of the Vth nerve, the nasociliary nerve, and the long ciliary nerves to the iris.

Fuchs[8] states that dilatation may be a spastic mydriasis often seen in states of cerebral irritation of the most varied sort or in stimulation of the cervical sympathetic, but that it is most commonly a paralytic phenomenon accompanying involvement of the oculomotor nerve, and apt, therefore, to be found in association with paralysis of accommodation in ophthalmoplegia interna or with oculomotor paralysis in ophthalmoplegia externa.

Table 3 is the record of pupillary dilatation.

TABLE 3 — PUPILLARY DILATATION.

Side	No. of Cases	Oculomotor paralyses						Ipsilateral complete ext. ophthalmoplegia	Sluggish light reaction	Absent light reaction	Orbital disease
		Ipsilateral		Contralateral		Bilateral					
		Complete oculomotor paralysis	Ptosis	Fixed contracted pupil	Ptosis	Superior rectus paralysis	Ptosis				
Bilateral	26						1		12	5	
Ipsilateral	10	1	4	7		1		1	2	2	1
Contralateral	7				1				1	1	
Total	*43*	*1*	*4*	*7*	*1*	*1*	*1*	*1*	*15*	*8*	*1*

Bilateral dilatation of the pupils occurred 26 times; unilateral dilatation was reported 17 times, 10 on the same side, seven on the opposite side.

Of 26 cases of bilateral dilatation, only one, a case without orbital disease, presented other evidence of oculomotor nerve involvement in the form of weakness of the levator palpebrae superioris of each side.

Light reaction was sluggish in 12 and absent in five.

A single lesion of the oculomotor nerves or the visual pathways themselves will not cause bilateral dilatation of the pupils, the finding present in approximately two-thirds of the

cases in which the state of the pupil was recorded; any possible etiology must necessarily be so diffuse and general in its effect that the lower visual pathways and centres of each side are equally involved and paralytic mydriasis is equally present in each eye. Increased intracranial pressure, probably on the oculomotor nerves, is such a factor and is responsible for pupillary dilatation in frontal lobe abscess.

A consideration of these facts brings the realization that bilateral dilatation of the pupils means increased intracranial pressure and nothing more.

A study of Table 3 shows that lesions of extrinsic ocular muscles occurred more frequently in ipsilateral than in contralateral or bilateral dilatation; when diffuse, uniformly increased intracranial pressure, as indicated by bilateral dilatation, was present, such lesions were infrequent.

Unilateral dilatation is slightly more frequent on the side of the cerebral abscess than on the side opposite the abscess and has, therefore, some lateralizing value, which is increased by association with lesions of extrinsic ocular muscles.

II. CONTRACTION.

Pupillary contraction may be caused, not only by spasm or the sphincter (spastic miosis), but also by paralysis of the dilator (paralytic miosis) of the iris; most authorities assign the greater importance to variations in the tone of the sphincter.

Sphincter contraction is effected through the superior colliculus, the pupillary centre in the anterior part of the Edinger-Westphal nucleus, and the oculomotor nerve; the superior colliculus receives stimuli from the ipsilateral optic tract (pupillary fibres from each retina), and from the ipsilateral visual cortex through the optic radiation.

Contraction may result, not only from light, but also from pressure on the visual pathways; Macewen[13] observed that the pupil is apt to be contracted when the cerebral abscess is small, dilated when it is large. Contraction also accompanies accommodation, a reflex which is integrated over an arc practically identical with that of light reaction; it also occurs in conjunction with convergence, but whether due to convergence or to the accompanying accommodation is a disputed point.

The sphincter of the iris is inhibited simultaneously with stimulation of the dilator through the cervical sympathetic by sensory stimuli from widely scattered skin areas, or by psychic stimuli from higher cerebral centres.

Fifteen observations on pupillary contraction were noted, one ipsilateral, two contralateral and 12 bilateral; only three of these 15 cases presented other evidence of oculomotor nerve involvement. Table 4 records the associated lesions in this group.

TABLE 4 — PUPILLARY CONTRACTION.

Side	No. of Cases	Oculomotor paralyses				Ipsilateral complete ext. ophthalmoplegia	Orbital disease
		Ipsilateral			Bilateral		
		Fixed dilated pupil	Internal rectus paralysis	Superior rectus paralysis	Superior rectus paralysis		
Bilateral	12		1	1			
Contralateral	2	2			1	1	1
Ipsilateral	1						
Total	*15*	*2*	*1*	*1*	*1*	*1*	*1*

SUMMARY OF PUPILLARY REACTIONS.

Bilateral pupillary reactions are the rule because the stimuli of which they are the result are bilaterally effective in the lower visual centres.

Bilateral reactions are indicative of increased intracranial pressure only and have no localizing value.

Unilateral reaction, especially when accompanied by other evidence of oculomotor nerve involvement, has some value in indicating the laterality of the cerebral lesion.

Dilated and contracted pupils are frequently accompanied by sluggish or absent light reaction.

D. OPTIC NERVE–I. VISION.

Observations regarding the vision were few in number and casual in character. The exact visual acuity was but rarely recorded; total blindness was occasionally noted.

Five cases presented impaired vision in the ipsilateral eye, one in the contralateral eye and 15 in both eyes; orbital disease was more frequently present in unilateral than in bilateral impairment. The incidence of bilateral visual impairment was the same in the cases with and without orbital complication.

Foster Kennedy,[11] in speaking of the effect on vision of a frontal lobe tumor (or abscess), makes the statement that when such a lesion is situated in the central part of the frontal lobe, direct pressure on the ipsilateral optic nerve is less apt to be present than is general increase of pressure, which causes bilateral papilloedema without visual impairment. When the lesion is situated in the basal part of the frontal lobe, direct downward pressure on the optic nerve may cause ipsilateral compression neuritis and impaired vision, followed by primary optic atrophy; accompanying cerebral edema may cause papilloedema with good vision in the opposite eye (basal frontal syndrome).

II. FUNDUS.

Unilateral fundus lesions, summarized in Table 5, occurred most frequently on the side of the cerebral abscess.

Bilateral fundus findings, without record of visual acuity, are recorded in Table 6; the greater involvement occurred most frequently on the side of the cerebral lesion.

Evidence of stasis, as manifested by congestion of retinal veins or by choked disc (papilloedema) was present in 84 per cent of the cases in which fundus notes were recorded; choked disc was present in 46 per cent (16 per cent of the entire group of 204 cases).

This small material indicates that fundus disease in abscess of the frontal lobe is due primarily to disturbance of circulation rather than to inflammation.

It is not within the province of this paper to discuss at length the mechanism of fundus lesions or to attempt an

evaluation of the factors in their production, inflammation, toxic or chemical irritants, or stasis within the sheath of the optic nerve, due to direct downward pressure or to interference with the circulation of the cerebrospinal fluid. The opinion is now quite generally held that papilloedema and choked disc are synonymous terms, and that optic neuritis introduces an inflammatory reaction, from whatever cause. Confusion may have been present, especially in the minds of the older

TABLE 5 — UNILATERAL FUNDUS LESIONS.

Lesion	Side	
	Ipsilateral	Contralateral
Congestion	3	1
Papilledema	2	
Choked disc	1	
Optic neuritis	5	1
Atrophy	1	
Total	*12*	*2*

TABLE 6 — BILATERAL FUNDUS LESIONS.

Lesion	No. of Cases	Most marked on	
		Ipsilateral side	Contralateral side
Congestion	14		
Papilledema	11	3	1
Choked disc	36	6	1
Optic neuritis	13	1	2
Atrophy	4	1	
Total	*78*	*11*	*4*

authors, with regard to the exact differentiation between optic neuritis and choked disc; Fuchs[8] remarks that the distinction is better established theoretically than in practice.

Benedict[4] states that the consensus of opinion is that frontal lobe abscess causes choked disc rather infrequently and has but little localizing value for the reason that the cerebral edema responsible for the disc changes is not always constant, or present only on the side of the abscess.

Lillie,[14] in a series of 11 cases of encapsulated frontal lobe abscess, observed bilateral choked discs, varying from 1 to 6 diopters, in eight cases; vision was good and fields revealed

only a mild concentric contraction for form and color. Halstead and Vaughan[19] found optic neuritis and choked disc frequently present and greater on the side of the lesion, due to direct pressure on the nerve.

Coleman[5] noted no relation between the size of the abscess and the degree of choking, inasmuch as the latter may be due more to encephalitis or massive cerebral edema than to the abscess.

III. VISUAL FIELDS.

Reports of careful visual field examinations were rarely encountered; fields normal on gross testing were but infrequently noted. It may, therefore, be of interest to refer to Eagleton's writings, which emphasize the value of field studies in the diagnosis of intracranial lesions.

Eagleton,[7] in his monograph on brain abscess, published in 1922, and in later writings,[6] dwells on the importance of cerebral edema in the production of disc and field changes. It is a factor constantly present in intradural suppurative lesions and varies much in extent and degree; the greatest edema is not necessarily on the side of the lesion. Edema may manifest its presence by uneven, transient, hemianoptic indentation of the contralateral field; the variability of the edema is reflected in changing fields which necessitate frequent examinations, the value of which is greatest when the diagnosis is as yet uncertain.

When the cerebral edema is generalized, as is the case when it has extended from the posterior fossa to the great brain, the fields usually show slight general contraction with hemianoptic tendency.

Lillie[14] observes that variations in cerebral edema, as indicated by changing discs and fields, point to an intradural process not yet stabilized (unencapsulated abscess); surgery is more apt to be satisfactory when encapsulation, indicated by stabile discs and fields, has been established.

Lillie,[14] Benedict[4] and others point out that symptoms of encapsulated abscess are those of tumor similarly situated and that abscess presenting tumor symptoms is most amenable to surgery.

E. PYRAMIDAL TRACT.

Pyramidal tract involvement, frequent in this study, was manifested by muscular twitching, general or local (Jacksonian attacks), or by weakness of muscle groups or body halves (paresis, paralysis, hemiparesis or hemiplegia).

The proximity of the cortical motor centres, the internal capsule, and the corticospinal and corticobulbar motor tracts to the posterior portions of the frontal lobe offers a ready explanation of these symptoms; the fact that frontal lobe lesions exert their effects on these pathways before decussasation explains the development of most pyramidal tract symptoms on the contralateral side of the body. Such symptoms on the ipsilateral side, more frequent in lesions of the posterior fossa, are due to dislocation of the medulla against the basilar plate of the occipital bone with resultant pressure on the pyramid of the opposite side above decussation, or to herniation of the medulla into the foramen magnum with pressure on the ipsilateral pyramidal tract below decussation, in either case with resultant ipsilateral symptoms. Such symptoms occur but rarely in supratentorial lesions; they may be caused by shifting of the brain with resultant pressure on the contralateral pyramid by the tentorium cerebelli. Meyers[16] suggests that physiological or anatomical variations in the pyramidal tracts, such as preponderance of ipsilateral fibres, or even absence of decussation, may be responsible for such findings.

Consideration of these facts precludes the possibility of attaching absolute lateralizing value to such signs; they may be highly suggestive but must be evaluated in connection with other factors present.

Generalized twitching, present in two cases, is indicative of widespread cortical irritation caused by increased intracranial pressure.

Ipsilateral localized twitching, significant of irritation of the opposite motor cortex, appeared more frequently than did contralateral twitching, indicative of cortical irritation on the side of the lesion.

Bilateral twitching was frequently present; it has no lateralizing or localizing value.

The incidence of unilateral and bilateral localized twitching is recorded in Table 7.

Generalized convulsions, indicative of increased intracranial pressure, were present in 59 cases, with loss of consciousness in 17. This finding has, of course, no localizing value.

Table 8 summarizes localizing symptoms which appeared in connection with general convulsions; such symptoms were more frequent on the contralateral side of the body, indicat-

TABLE 7 — LOCALIZED TWITCHING.

Site	Side		
	Ipsi-lateral	Contra-lateral	Bi-lateral
Face	2	1	3
Arm	4	2	3
Hand		1	1
Leg	3		2
Total	9	4	9

TABLE 8 — GENERALIZED CONVULSIONS.

Beginning in:	Side	
	Ipsi-lateral	Contra-lateral
Face	1	2
Arm		1
Hand		1
Total	1	4
More marked on one side of body	1	4

ing cortical irritation on the side of the lesion. Ballance[2] makes the statement that the initial local spasm of a fit or a local paresis is pathognomonic of the site of the lesion.

The etiology and localizing value of Jacksonian attacks does not need statement here; a study of Table 9 shows that, while contralateral extremities were involved more frequently than those of the ipsilateral side, the two sides of the face were equally involved. The tendency of disturbances of facial musculature to appear with equal frequency on the two sides has been noted elsewhere.

The occurrence of contralateral and bilateral Jacksonian attacks in cases of solitary cerebral abscess necessitates caution in the interpretation of unilateral manifestations.

Table 10 illustrates very well the decided contralaterality of lesions characterized by impairment or loss of muscular function; the incidence of such lesions on the two sides of the face is much more nearly the same than that of lesions occurring elsewhere.

TABLE 9 — JACKSONIAN ATTACKS.

Site given	Side		
	Ipsi-lateral	Contra-lateral	Bi-lateral
Face	2	2	1
Arm	3	7	4
Leg	1	3	5
Total	*6*	*12*	*10*
Site not given	1	10	

TABLE 10 —
A. Paresis and Paralysis.

Site	Side	
	Ipsi-lateral	Contra-lateral
Face	15	19
Arm	2	34
Hand	1	8
Leg	1	20
Total	*19*	*81*

B. Hemiparesis and Hemiplegia.

Side	
Ipsilateral	Contralateral
3	31

The occurrence of paresis or paralysis on either side of the body lessens the lateralizing value of all unilateral findings.

SUMMARY OF PYRAMIDAL TRACT SYMPTOMS.

The significance of symptoms dependent upon involvement of the motor cortex and tracts may be summarized by saying that general convulsions and generalized twitching have no lateralizing or localizing value; they mean only increased intracranial pressure. Unilateral Jacksonian attacks have definite lateralizing and localizing value, pointing directly to the precentral gyrus; when bilateral they mean only increased

pressure. Partial or complete loss of muscular power in one member or one-half of the body has both lateralizing and localizing value; when the muscular system of half the body is involved an extensive lesion of the precentral gyrus or a localized lesion in the internal capsule or motor tracts is indicated.

An attempt was made to associate the site of the lesion with the symptoms present; this was impossible in most cases, owing to the lack of precise information concerning the exact location of the abscess; such information was occasionally given, but in the majority of cases the findings at operation or autopsy were not reported accurately enough to be so used.

F. MOTOR CRANIAL NERVES NOT ELSEWHERE DISCUSSED.

The nuclei of the cranial nerves have a bilateral crossed cortical connection which is effected through the pyramidal tracts, principally that of the opposite side; the variability of these pathways, commented on in discussing their lesions, is reflected in the variable cortical representation of cranial nerves.

Irritative symptoms, due to stimulation of the motor cortex, appear on the opposite side of the body; paralytic lesions, if central (above the decussation of the pyramids) also appear on the opposite side of the body, as do the symptoms of pyramidal tract involvement with which they are frequently associated; paralytic lesions, caused by direct or transmitted pressure on the peripheral nerve, appear on the side of the pressure. Unilateral paralysis as the result of an upper motor neuron (central) lesion is rendered unlikely by the bilateral cortical connection of the cranial nuclei; most such lesions are peripheral.

A glance at Table 11 shows that the facial nerve was most frequently involved. Irritative symptoms appeared with equal frequency on the two sides of the face; paralytic lesions were slightly more frequent on the contralateral side. This was true, not only of the facial nerve, but also of the motor division of the trigeminal and of the hypoglossal.

Bilateral symptoms, such as pharyngeal paresis and laryngeal spasm, were undoubtedly caused by pressure on bulbar nuclei.

Pressure of the cerebral abscess itself may cause peripheral paralytic lesions on the ipsilateral side; such lesions on the contralateral side can be explained only on the basis of pressure from cerebral edema.

Variations in the pressure of cerebral edema offer a reasonable explanation for the more frequent occurrence of paralytic lesions of the trigeminal, facial and hypoglossal nerves on the contralateral instead of the ipsilateral side, as was the case in lesions of the oculomotor, trochlear and abducens

TABLE 11 — MOTOR CRANIAL NERVES NOT HITHERTO CONSIDERED.

Nerve	Paralysis			Twitching			Spasm			Jacksonian Attacks		
	Ipsilateral	Contralateral	Bilateral	Ipsilateral	Contralateral	Bilateral	Ipsilateral	Contralateral	Bilateral	Ipsilateral	Contralateral	Bilateral
Trigeminal (mandibular branch		1[a]										
Facial	15	19		2	2	6				4	4	1
Vagus			4[b]						1[c]			
Accessory												
Hypoglossal	2[d]	4[d]										
Total	*17*	*24*	*4*	*2*	*2*	*6*			*1*	*4*	*4*	*1*

a. Paralysis of pterygoid muscle with jaw deviation b. Paralysis of pharyngeal constrictors
c. Laryngeal spasm d. Paralysis of tongue

nerves. Such variations but emphasize the fact that any lateralizing significance possessed by unilateral lesions of cranial nerves is but limited.

G. SENSORY DISTURBANCES.

The literature on disturbance of sensation in frontal lobe abscess is scant and but few notes concerning it were found in the case reports examined.

Cortical sensory representation is, like cortical motor representation, largely crossed; afferent sensory tracts, whether

those of pain, temperature, light touch or discriminative touch, decussate almost completely, if not entirely, before leaving the upper level of the medulla. Symptoms caused by a lesion above the medulla, such as abscess of the frontal lobe, are on the opposite side of the body. It is only reasonable to assume that here, as in the case of ipsilateral pyramidal tract signs, direct fibres may occasionally be present in unusually large numbers and so account for ipsilateral sensory disturbances.

The mechanism responsible for sensory disturbances is that involved in the production of motor symptoms, *viz.*, transmitted or direct pressure, or actual involvement of the cortical centres or fibre tracts entering or leaving the internal capsule; the location of the cortical sensory area in the postcentral gyrus lessens the incidence of sensory as compared with motor symptoms.

Ballance[2] observes, in discussing frontal lobe tumor, that there is no loss of common sensation on the opposite side of the body when the sensory cortex is involved, but that there is loss of muscle sense and of the power of localizing light touch.

Osnato[19] mentions as symptoms due to involvement of the sensory cortex disabilities connected with the recognition of size, form and shape of objects, and disturbances in deep muscle sensibility and in discrimination between one and two points which are ordinarily recognized at 3 mm. or less; in frontal lobe abscess such points may not be recognized at much wider spacing.

Anesthesia of the ipsilateral face was present in one case, hyperesthesia of the contralateral face and cornea in one case. Bilateral corneal anesthesia was noted once. Impaired sensation of the opposite side of the body was found twice, and numbness of the ipsilateral side once. Disturbed sensation was present once in each arm.

Anosmia, due to direct downward pressure on the olfactory nerve, is a not uncommon symptom in both frontal lobe tumor and abscess. In this series of cases it was noted twice. It doubtless would be found more frequently by careful examination.

This material, too scanty to be of value, suggests the advisability of looking for disturbances of sensation in frontal lobe abscess cases whenever possible.

H. REFLEXES.

The notes on reflexes in this series of cases were disappointing because of brevity and incompleteness. In many cases there was only a casual observation on one reflex, giving the impression of a hurried and incomplete neurological examination. In a few cases a complete examination had been made and the findings recorded; in still fewer instances were there progress notes on the variations in the reflexes.

Reflex mechanism is complicated, depending on spinal cord pathways, which are, in many cases, known in detail, and on extensive ramifications, both in cord and cortex, which are not so well known. The study of reflexes may be expected to yield information concerning the integrity of the spinal reflex arc, its connections with the cortex, and the control which the cortex exercises upon the lower centres.

Superficial reflexes are phylogenetically younger than deep reflexes and are dependent on normal cortical activity. Increased cortical activity or tone in the neuromuscular mechanism increases superficial reflexes; lowered activity or tone may cause their disappearance. Any lesion which breaks the spinal arc in the anterior horn cell, efferent motor nerve, afferent sensory nerve, or in the posterior root, will obviously ablate these reflexes, as will a lesion of the upper motor neuron, presumably by removal of the activating action of the cortex. The pathways through the cortex are not as well understood as are the spinal arcs; the pyramidal fibres pursue a well known course but there are probably tracts from the premotor area with important connections, whose course is not well known.

The deep reflexes are phylogenetically older than the superficial and are controlled in part by the inhibitory action of the cortex. Lesions of the upper motor neuron remove this inhibitory action and cause hyperactive deep reflexes; lesions of the spinal arc or posterior columns may destroy them entirely. Numerous deep reflexes, such as the ankle clonus or Babinski, are in reality the manifestations of increased neuromuscular

tone due to removal of cortical inhibition, or to meningeal or posterior root irritation.

Superficial and deep reflexes are profoundly affected by the state of mind and body and reflect accurately conditions such as increased mental activity or shock; under such circumstances reflexes have no localizing value. When discrepancies appear between the superficial and deep reflexes, or between reflexes of opposite sides or of different levels, or when abnormal reactions are persistently present, the finding assumes value.

TABLE 12 — SUPERFICIAL REFLEXES.

Abdominal Reflex	No. of Cases	Accompanying Reflexes						
		Hyperactive cremasterics	Kernig's sign present	Achilles' jerk present	Babinski's sign present	Ankle clonus present	Knee-jerks hyperactive	Knee-jerks absent
Hyperactive								
Contralateral	1	1	1	1				1
Bilateral	3							
Absent								
Ipsilateral	1		3	2	2	2	1	3
Contralateral	1							
Bilateral	6							
Total	*14*	*1*	*4*	*3*	*2*	*2*	*1*	*4*

Abscess of the frontal lobe affects superficial and deep reflexes by disturbing their cortical associations through generalized pressure or actual destruction of nerve tissue.

Reflex changes noted in this study were almost entirely bilateral; only two were unilaterally present in numbers sufficient to give them any importance. There were many isolated findings unrelated to anything else of diagnostic importance; manifestly they were of no value.

Table 12 records the notes on superficial reflexes.

Hyperactive superficial reflexes are due to increased cortical activity or irritability of the neuromuscular system; this was

borne out, in this small group, by the hyperactive cremasterics and the positive Kernig, the latter indicative of meningeal irritation, especially about the posterior roots.

The abdominal reflexes were bilaterally absent in eight cases and unilaterally absent once on each side; the accompanying reflexes were indicative, in almost every case, of upper motor neuron impairment. Babinski's sign, the Achilles jerk, and the ankle clonus were each present twice in this small group; Kernig's sign was positive three times; the knee jerks were hyperactive once and absent three times. These findings suggest a serious lesion, either meningeal or of the upper motor neuron.

TABLE 13 — BILATERAL DEEP REFLEXES.

Achilles' jerk	4
Ankle clonus	8
Babinski's sign	7
Knee-jerks hyperactive	10
Knee-jerks absent	8
Kernig's sign	24

TABLE 14 — UNILATERAL DEEP REFLEXES.

Reflex	Side	
	Ipsilateral	Contralateral
Ankle clonus	2	7
Babinski's sign	2	5

Changes in the deep reflexes, summarized in Table 13, were more commonly noted. Bilateral Achilles' jerk was present four times; the accompanying deep reflexes indicated either meningeal irritation or upper motor neuron disturbance. Ankle clonus was present eight times, Babinski's sign seven times, hyperactive knee jerks, 10 times; knee jerks were absent eight times. These findings were bilateral and associated with various combinations of deep reflexes which were without particular pattern.

Kernig's sign was present in 24 cases, being the reflex most frequently reported.

Unilateral deep reflexes (Table 14) were reported infrequently; crossed motor and sensory innervation explains their more frequent occurrence on the contralateral side.

Spasticity was reported once in each arm and once in both arms; it was present once in the ipsilateral leg and twice in both legs.

Loss of control of bowel and bladder sphincters was noted in 10 cases; in five, loss of bladder control was present without bowel disturbance. These findings have the same general significance as that attributed to the presence of the deep reflexes noted above.

SUMMARY OF REFLEX CHANGES.

The bilateral reflex changes noted in this group of frontal lobe abscesses were without lateralizing or localizing value; they indicated meningeal irritation or ablation of upper motor and sensory neuron function. Unilateral changes occurred most frequently on the side opposite the lesion.

EQUILIBRIUM AND CO-ORDINATION.

Disturbances in equilibrium and co-ordination were but rarely noted, either in the case histories or literature studied. Staggering gait before operation was noted in one case; in one an ataxia, otherwise undescribed, was present on both sides of the body. In one case an ataxia was present only on the side contralateral to the lesion.

Nystagmus directed to both sides was present in four cases, to the contralateral side only, in one case. Adiadokokinesis was present on the contralateral side in one case. These findings comprise the entire list of such disturbances in this series of cases.

Frontal ataxia has been described by various writers. Ballance[2] states that in this condition the patient sways to either side when standing and would fall if not supported; in walking he does not have the zigzag staggering gait of cerebellar lesions. The ataxia is attributed to lack of attention or possibly paresis of the muscles of the trunk and neck. Osnato[19] and Wechsler[23] attribute this symptom to disturbance of the frontopontocerebellar and frontoparietal-occipital tracts con-

necting the frontal lobe with the opposite cerebellar hemisphere. In man there is a large tract from the frontal cortex directly to the pons; from here fibres carry the impulses to the opposite cerebellar hemisphere via the middle cerebellar peduncles. This is so well developed that it is surprising that symptoms are not more frequent in extensive frontal lesions.

Foster Kennedy[11] makes the statement that the only posture characteristic of such disease (frontal lobe tumor) is titubation on the heels and a tendency to retropulsion. Such disturbances are important only in that they may simulate a cerebellar lesion.

Nystagmus was but rarely mentioned; it was present in five cases, in four of which it was directed to both sides. Grinker[9] states that the communication between the cranial cavity and internal ear permits the transmission of variations of intracranial pressure to the latter organ, a very possible cause for the symptom. It may also be due to pressure on the vestibular nerve, tracts or nuclei.

I. APHASIA.

Twenty-nine cases presented speech difficulties. In three the condition was termed aphasia, in one sensory aphasia without agraphia, and in one paraphasia with word blindness; in the others the speech was described as impaired, slurred, slow or lost.

Aphasia is described by Wechsler[23] as "a disturbance in the highest speech mechanism, in the realm of speech concepts, in the sphere of symbolic thinking. In aphasia the formulation of propositions and their relations is impaired or lost. The disturbance is in the comprehension, elaboration and expression of speech concepts." Older views held that speech was a higher faculty capable of dissociation by disease into lesser faculties, each located in a more or less definite anatomical centre, destruction of which caused the so-called pure aphasias, such as visual or auditory aphasia, sensory or motor agraphia.

Recent work, notably that of Head (quoted by Wechsler[23]), indicates that the mechanism of aphasia is not as simple as was at first believed. The newer concept of aphasia, as defined by Wechsler, involves the idea that speech is a highly intel-

lectual process, the "symbolic thinking" of Head, which breaks down, not into subfaculties or pure aphasias, but into broader, more inclusive concepts of speech comprehension and expression, not located in definite anatomical centres. Aphasia in any of its forms affects the whole intellectual process, even though a particular concept is especially involved.

On the basis of this theory several types of aphasia are recognized. Verbal defects, characterized by defective power of forming words, either for internal or external use, with intact comprehension, correspond to the motor or expressive aphasia of the older terminology. Syntactic defects are disorders in the balance and rhythm of symbolic expression, comparable to word deafness. Nominal defects are difficulties in the designation of objects by name (global aphasia); the defects in writing and reading here involved bear the older terminology of agraphia and alexia. Semantic defects involve difficulties in the comprehension of words and phrases as a whole (Head, quoted by Grinker[9]).

While the theory of definite centres for so-called pure aphasias is no longer universally accepted, clinical experience teaches that lesions in certain areas produce certain types of speech disturbance, which may, therefore, have localizing value. It is thought that the speech zone is in the cerebral hemisphere opposite the dominant hand. According to Wechsler,[23] left frontal lesions situated low in the pre-Rolandic area disturb spontaneous speech and expression of language; when such defects are associated with Jacksonian epilepsy or muscular disturbance of the opposite side, the pre-Rolandic area is still more definitely indicated as the site of the lesion. When comprehension of speech is mainly involved, the lesion is generally behind the Rolandic and below the Sylvian fissure, the upper part of the temporal lobe and the angular gyrus.

In this group of cases speech difficulty was observed in 29, but the designation impaired, slurred, slowed or lost does not fully describe the difficulty; it must be remembered that very sick patients frequently have speech difficulties, due to weakness and mental confusion, that are not true aphasias. Lesions of the frontal lobe are early characterized by inattention and lack of interest; drowsiness and apathy, attendant on the development of increased intracranial pressure, further

complicate the mental picture (Grinker[9]). These factors may so interfere with speech as to simulate an aphasia which cannot be ruled out without comprehensive neurological examination.

The reduction of increased intracranial pressure by the injection of hypertonic salt solution or by ventricular drainage may for a time relieve the mental symptoms due to pressure and allow the disturbances caused by the lesion itself to stand out more clearly.

In these 29 cases of speech disturbance the abscess was in the left lobe in 15 cases and in the right in 14; approximately two-thirds of each group gave evidence, in the form of Jacksonian attacks, paresis or paralysis, of involvement of the ipsilateral motor cortex. Only two of the 14 right-sided abscesses were found in the motor area; of the 15 left-sided abscesses seven were located in the motor area (46 per cent as contrasted with 14 per cent in right-sided lesions).

It is more than probable that some of the speech difficulties observed in both right- and left-sided lesions were not true aphasias, but abnormalities of expression due to other conditions, such as shock or toxemia.

The most exact localizing information derived from the presence of aphasia is found in cases in which it is associated with clear-cut Jacksonian attacks on the side of the dominant hand; under such circumstances the lower portion of the precentral gyrus on the side opposite the dominant hand is almost certainly involved.

J. PSYCHIC CHANGES.

The frontal lobes, particularly the left, are thought to be the seat of the higher psychic functions of the brain and their lesions are commonly accompanied by disturbances in the mental and psychic life of the patient. Ballance[3] notes, in subacute or chronic frontal lobe abscess, evidence of psychic dissolution with loss of the highest functions of the brain, such as ideation, memory, control, attention and judgment. Foster Kennedy,[11] speaking of frontal tumors, considers mental changes one of the most reliable guides; he enumerates lessened power of attention, irrelevant replies, trivial and

meaningless jocosity (Witzelsucht), inept and causeless laughter, hypersensitiveness to offense, effusive apologies for trifles, petit mal with sudden attacks of mental confusion, and hebetude, from which the patient is aroused with difficulty and which later passes into stupor.

Osnato[19] remarks on the presence of euphoric states, characterized by flippant and discordant emotional reaction in the direction of well being, a reaction out of proportion to the seriousness of the illness. Carelessness in dress and personal habits and inattention to and loss of interest in their business, home, or even their own illness, may not infrequently be seen in patients with frontal lobe abscess. Changes in personality are common; a patient naturally polite, co-operative and obedient becomes rude, obstreperous and unruly. Obscenity and profanity may be indulged in.

Most of these mental and psychic changes were present in this series of cases. Mental sluggishness was the most common; it was noted in 21 cases. Loss of memory was present in 14, and personality changes in 20. In one of my own patients the only thing indicative of disturbed intelligence was the momentary inability to remember his own name when entering the hospital; it did not appear again during the entire illness.

Such mental and psychic disturbances, while suggestive, possess no localizing value.

SUMMARY.

The results of an analysis of neurological symptoms noted in 204 cases of solitary abscess of the frontal lobe may be summarized as follows:

A: Neurological symptoms of frontal lobe abscess may be divided into two groups. In one group, composed of symptoms indicative only of increased intracranial pressure and, therefore, without localizing value, are bilateral and generalized manifestations; in the other group are symptoms caused by involvement of centres and tracts in or near the frontal lobe, neighborhood symptoms which have, therefore, some lateralizing and localizing value. Most unilateral and localized manifestations belong in this group.

In the first group are included the following ocular lesions, all bilateral: paralysis of extrinsic muscles, pupillary changes, visual impairment, and retinal, disc and field changes.

Also included in this group are bilateral and generalized twitchings, convulsions and paralyses.

Most reflexes, both superficial and deep, belong in this group; psychic and mental changes are also included.

In the second group, composed of symptoms which may have some lateralizing or localizing value, are unilateral lesions of extrinsic ocular muscles, conjugate deviation of the eyes, unilateral pupillary changes, unilateral disturbances in vision, retina, optic nerve and visual field.

Unilateral or localized twitching, spasm or paresis is of value; Jacksonian attacks, especially when associated with aphasia, may be of definite localizing value, as may hemiparesis and hemiplegia.

Unilateral reflexes, especially when persistently or repeatedly present, are of value in suggesting the side of the lesion.

Aphasia, especially when associated with Jacksonian attacks, has definite localizing value.

B: Symptoms of frontal lobe abscess may be ipsilateral, contralateral or bilateral.

Unilateral symptoms occurred more frequently on the side of the cerebral abscess than on the side opposite the abscess; they are, therefore, of some lateralizing value.

The frequent occurrence of unilateral symptoms on the side opposite the cerebral abscess limits their lateralizing and localizing value and necessitates caution in their interpretation.

Bilateral or generalized symptoms are indicative only of increased intracranial pressure.

CONCLUSIONS.

1. There is no neurological picture which is pathognomonic of frontal lobe abscess.

2. Neurological signs occur in frontal lobe abscess with sufficient frequency and possess sufficient localizing value to be of definite and, at times, considerable assistance, not only in making the diagnosis but also in localizing the lesion.

BIBLIOGRAPHY.

1. ATKINSON, E. M.: Abscess of the Brain: Its Pathology, Diagnosis, and Treatment. *Lancet*, 214:483, March 10, 1928.

2. BALLANCE, C.: Some Points in the Surgery of the Brain. London, Macmillan & Co., 1908.

3. BALLANCE, C.: A Glimpse into the Surgery of Abscess of the Brain. *Practitioner*, 119:1, July, 1927.

4. BENEDICT, W. L.: Abscess of the Brain from the Standpoint of the Ophthalmologist: Symposium Brain Abscess. *Tr. Am. Acad. Ophthal. and Otolaryngol.*, p. 52, 1929.

5. COLEMAN, C. C.: Brain Abscess: A Review of Twenty-eight Cases with Comment on the Ophthalmologic Observations. *Jour. Amer. Med. Assn.*, 95:568, Aug. 23, 1930.

6. EAGLETON, W. P.: Localizing Value of Ophthalmic Examinations in Suppurative Diseases of the Brain. *Jour. Amer. Med. Assn.*, 92:713, March 2, 1929.

7. EAGLETON, W. P.: Brain Abscess, New York, MacMillan, Co., 1922.

8. FUCHS, E.: Diseases of the Eye, ed. 10, translated by E. V. L. Brown, Philadelphia, J. B. Lippincott Co., 1933.

9. GRINKER, R. R.: Neurology, ed. 1. Springfield, Thomas, 1934.

10. HALSTEAD, A. E., and VAUGHAN, R. T.: Frontal Lobe Abscess. *Jour. Mich. State Med. Soc.*, 11:73, Feb., 1912.

11. KENNEDY, F.: The Symptomatolgy of Frontal and Temperosphenoidal Tumors. *Jour. Amer. Med. Assn.*, 98:864, March 12, 1932.

12. LEEGAARD, F.: Cerebral Abscess of the Frontal Lobe Originating from the Frontal Sinus and Other Intracranial Complications Resulting from Inflammatory Processes of the Nasal Accessory Sinuses. *Ann. Otol., Rhinol. and Laryng.*, 28:95, March, 1919.

13. MACEWEN, W.: Pyogenic Infective Diseases of the Brain and Spinal Cord. Glasgow, James Maclehose & Sons, 1893.

14. LILLIE, W. I.: The Clinical Significance of Choked Discs Produced by Abscess of the Brain. *Surg., Gynec. and Obst.*, 47:405, Sept., 1928.

15. MEYERS, I. L.: Conjugate Deviation of the Head and Eyes. *Arch. Otolaryngol.*, 13:683, May, 1931.

16. MEYERS, I. L.: Brain Lesions with Homolateral Phenomena of Pyramidal Tract Involvement. *Calif. and West. Med.*, 34:354, May, 1931.

17. NEUMANN, H.: Abscess of the Brain. *Jour. Laryng. and Otol.*, 45:377, June, 1930.

18. OSLER, W.: Modern Medicine, ed. 3, edited by Thos. McCrae. Philadelphia, Lea & Febiger, vol. 6, p. 357, 1928.

19. OSNATO, M.: Discussion to Imperatori: Brain Abscess (Frontal Lobe) Complicating Frontal Sinusitis; and to Hastings: Osteomyelitis of the Frontal Sinus. *Tr. Am. Laryng. Assn.*, p. 53, 1927.

20. RILEY, H. A.: The Central Nervous System Control of the Ocular Movements and the Disturbance of This Mechanism. *Arch. Ophthal.*, 4:640, Nov., 1930, and 4:885, Dec., 1930.

21. SKILLERN, R. H.: Discussion to Berry: Brain Abscess of Paranasal Sinus Origin. *Tr. Am. Laryng. Assn.*, p. 194, 1923.

22. SPIEGEL, E. A.: Role of Vestibular Nuclei in the Cortical Innervation of the Eye Muscles. *Arch. Neurol. and Psychiat.*, 29:1084, May, 1933.

23. WECHSLER, I. S.: A Textbook of Clinical Neurology, ed. 2. Philadelphia, W. B. Saunders, 1932.

24. WHITNALL, S. E.: The Anatomy of the Human Orbit and Accessory Organs of Vision. London, Henry Frowde and Hodder and Stoughton, 1921.

1371 Lowry Building.

AN UNUSUALLY LARGE CALCULUS OF THE TONSIL.*

DR. HERMAN RUBIN, Brooklyn.

A man, age 32 years, was admitted Sept. 26, 1933. During the past three months he had complained of an unproductive cough, general weakness and insomnia. He had lost about 15 pounds during this time. For the past month or so he had been slightly hoarse.

Examination of the chest revealed exaggerated breath sounds with large rales in the right apex. Few rales were heard on the left side. The X-ray report of the chest indicated acinous exudative and productive changes in both upper lobes. The process was slightly more extensive on the right side. No definite cavitations were seen. Examination of three specimens of the sputum were negative for the presence of tubercle bacilli and fungi.

For several months he had noticed a lump in the right side of his throat which had gradually become larger and which had caused more or less constant pain in this region. The pain was aggravated by swallowing and he experienced the sensation of a foreign body in his throat.

On examination a large mass about the size of a small plum was seen in the right tonsillar fossa. This grayish-white mass occupied the usual position of the tonsil and was surrounded by a thin border of normal tissue all around. It was of bony consistency, freely movable and was held within the tonsillar pillars. Palpation revealed the mass to be of bony hardness. Examination with the probe produced a grating sound. A diagnosis of calculus was made.

A lateral X-ray of the neck showed the presence of a large dense, oval mass, measuring about 1½ in. x 1 in. in diameter, in the region of the angle of the jaw. This was considered as a probable calculus in the tonsil. Smears taken from the tonsillar area showed the presence of fusiform bacilli and occasional spirochetes. No tubercle bacilli were found.

---

*From the Otolaryngological Service of Dr. M. C. Myerson, Sea View Hospital, Staten Island, N. Y.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 3, 1936.

The tonsil with its stone embedded was removed by dissection and snare, under local anesthesia. The usual tonsil snare loop was much too small to encircle this mass. Examination of the specimen showed tonsil tissue to be present only on the posterior and lateral surfaces. On section, the tissue gave no evidence of tuberculosis. The tonsil weighed 24-2/3 gm. (370 gr.) and measured 37 x 28 mm. This calculus was oval in shape, smooth over most of the surface area, with scattered, fine linear markings as is seen in coral formation. One surface was excavated, presenting a smoothly polished and faceted concavity approximately 3 mm. in depth.

Fig. 1. Lateral Roentgenogram illustrating large calculus in the tonsillar region.

Calculi of the tonsils are spoken of variously as stones, concretions and tonsilloliths. Weller[6] found 8 per cent of the calcareous deposits in a series of 1,000 cases which were examined histologically. He states that these rarely give rise to symptoms and are found when bisecting the tonsil. These stones may be visible at the mouth of an enormously dilated crypt or beneath an area of ulceration and are prone to recur when simply removed. Tonsillectomy is the only assurance against recurrence. They may arise in the tonsillar crypts around colonies of organisms or keratohylin masses.

Gibb[1] thought that the cause of the tonsillolith in his case was due to the healing of a tuberculous deposit in the tonsil.

Shah[4] reported a case in which after incision into what resembled a tonsillar abscess, a concretion about the size of a small betel nut weighing 80 gr. was removed. Goodman[2]

when applying silver nitrate to what he thought to be an ulcer of the tonsil, in a male, age 20 years, discovered a tonsillar stone weighing 50 gr. (3.3 gm.)

Woodman[7] reported a case in a male, age 64 years, who complained of a sensation of a foreign body in his mouth, which was causing dysphagia and was accompanied by an abscess in the upper part of the right side of the neck. The sinus resulting from this abscess healed after the removal of a stone which weighed 3½ gm.

Fig. 2. Actual size of calculus.

Swain[5] removed a calculus in a lady, age 72 years, from whose tonsil he had previously removed a calculus 15 years before. Fourteen years prior to this, she had had another stone removed. She had had calculi in the same tonsil on four different occasions.

One of the largest size stones recorded in the literature was reported by Robertson[3] in a man, age 50 years, who had had frequent attacks of tonsillitis. He finally developed a suppurative process and nearly choked because of the foreign body in his throat. He expelled a large oval stone about the size of a small hen egg during a violent coughing spell. This calculus weighed 1 oz. (30 gm.)

CONCLUSIONS.

It is unusual to encounter stones of such large proportion in the tonsil today. This is so, principally, because the patient, seeking relief, would consult the laryngologist early.

This case is reported as the incidental finding of an unusually large calculus in a tuberculous individual.

BIBLIOGRAPHY.

1. Gibb, W.: Calculus from the Tonsil. *Trans. Path. Soc.*, 11:106, London, 1860.

2. Goodman, H. S.: Calculus in the Tonsil. The Laryngoscope, 40:290, April, 1930.

3. Robertson, W. G. A.: Large Tonsillar Calculus. *Brit. Med. Jour.* 1:14-16, 1899.

4. Shah, M. R.: Calculus in the Tonsil. *Indian Med. Gaz.*, 66:199-200, April, 1931.

5. Swain, H. L.: Recurrent Calculus of the Tonsil. *Ann. Otol., Rhinol. and Laryngol.*, 29:73, March, 1920.

6. Weller, C. B.: Incidence and Pathogenesis of Tonsillar Concretions. *Ann. Otol., Rhinol. and Laryngol.*, 33:79-119, March, 1924.

7. Woodman, G. S.: A Case of Tonsillar Calculus of Unusual Size. *Brit. Jour. Surg.*, 8:375-376, 1920-1921.

277 Eastern Parkway.

PRELIMINARY REPORT ON AN OPERATION FOR THE RELIEF OF DOUBLE ABDUCTOR PARALYSIS.*

DR. JOHN M. LORE, New York.

In this report I do not want to give the impression that it is the answer to the problem of double abductor paralysis; this is an anatomical study. It may not be worth anything but we have had a lot of fun in doing it. We do know that other procedures have failed.

It is very interesting when you go back into the history of this subject to find that the veterinarians have been doing a lot of this work, and some of the procedures that have been used on horses have been tried on the human and given up. The procedures that have been applicable to horses have not been applicable to humans because of anatomical differences.

What happens in the larynx when a biopsy is done? There is regeneration of the cord and replacement of the tissues removed. In the cat work I have done, we have been able to show that if you remove part of the cord there is a filling in of the gap with granulation tissue. For a while that granulation tissue will grow beyond the midline. In the meantime epithelial tissue will be growing in from the sides. When it is completely epithelialized, you have a new cord with straightening of the deformity. The same thing happens in laryngofissure. (Demonstration of pictures.) In the operation of laryngofissure in which considerable tissue is removed, we know there is a formation of granulation tissue to fill in the gap. Sometimes it gets to a point beyond the cord and we wonder if there has been a recurrence of the growth. Sometimes we have to take out some of it and send it to the laboratory. More often than not the report comes back, "granulation tissue." In the meantime epithelium grows in and bridges over the gap. We then get this type of picture. (Demonstration.) While these forces are acting we begin to find that

*Read before the New York Academy of Medicine, Section on Oto-Laryngology, Nov. 20, 1935.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Feb. 15, 1936.

there is a straightening out of that curved line and of the protrusion in the midline. Instead, we get this other picture showing the cord from the anterior commissure to the arytenoid, if it is still there, or the place where it had been, simulating a true cord so that it is sometimes difficult to tell whether it is a true or false cord. The two fixed points of the cord are the anterior commissure and the arytenoid, and the pull exerted between these two points tends to keep the cord straight. What would happen if we removed one of these points and transferred it to a point where the cord would be more external? That is the basis of this whole procedure.

The interior of the larynx was exposed by means of a thyrotomy incision, extended into the thyrohyoid membrane in the form of a V-shaped incision. Then a small bladed knife was plunged through the floor of the ventricle, about three-eighths of an inch from the edge of the cord, and coming out about three-eighths of an inch subglottically. The knife, in this position, was carried posteriorly towards the arytenoid, cutting parallel to the vocal cord. When the vocal process was reached, the knife was carried internal to it, cutting the attachment of the vocal cord from it, until the posterior commissure was reached, at which point the cord was severed. The arytenoid was then dissected and removed. The tissue subjacent to the detached cord was removed down to, but not including, the perichondrium. The vocal cord was then put back in its original position and anchored to the surrounding tissues by means of sutures.

The result when we suture this tissue is to transfer the posterior end of the cord attachment externally. This may be kept in place by packing inside the larynx or perhaps by a bag inflated with air. This afternoon I performed this operation on the cadaver. I do not believe everyone saw it and therefore, I brought this specimen, if you should care to examine it. In this specimen the arytenoid has been removed and the cord has been transferred externally. You can see when you close the larynx the amount of space it creates. On the cadaver the operating time after the larynx has been entered is about 12 minutes, but in the human it would be longer because of the bleeding.

Technically, it is a very simple procedure. It is in no way comparable to the technique involved in the so-called submu-

cous resection. I had tried that long before it was reported by Dr. Hoover and had given it up. I can see some problems in connection with this procedure; I do not know how this will work out. Perhaps when I report this work at the January meeting I will have some more points. I am submitting this preliminary report for your constructive criticism, and anything you have to offer in the way of criticism will certainly be appreciated.

325 West End Avenue.

THE AMERICAN BOARD OF OTOLARYNGOLOGY.

An examination was held in Kansas City, May 9, 1936, during the meeting of the American Medical Association. Forty-three candidates were examined, of which number 34 were passed and nine were conditioned or failed.

The Board desires to announce that hereafter examination will be of two days' duration — including written as well as oral tests. Furthermore, after Jan. 1, 1938, no certificate will be granted "formally," that is, without examination and all candidates shall take the pathology examination.

An examination will be held in New York City, Sept. 25 and 26, just prior to the meeting of the American Academy of Ophthalmology and Otolaryngology. Prospective applicants for certificate should secure application blanks from the Secretary, Dr. W. P. Wherry, 1500 Medical Arts building, Omaha, Nebr.

A NEW DEVICE FOR TESTING MALINGERING IN UNILATERAL DEAFNESS.

DR. M. MARTYN KAFKA, Brooklyn.

The otologist may be called upon on different occasions to give testimony whether or not the statement or claim of a patient is justified in a so-called unilateral deafness which is attributable to trauma. It may necessitate the proof that the patient is trying to glean sympathy from those about him, or that he is attempting to avoid his obligations of performing his duties; on the other hand, the individual who presents such claims may be entirely swayed by the idea of financial remuneration. The specialist must decide, at times, in a court room what precise state is actually present.

Malingering for financial return is the most usual type encountered, as a general rule, and in such instances there must not be any particular confusion with such mental types as found in psychotic patients.

In order to more completely understand the mechanism existing in certain psychotic reactions, the examiner must remember that a psychotic may possibly simulate, just as any other type.

The otolaryngolist may also find types of individuals artfully setting forth claims, but who have had pre-existing chronic otitis media of purulent state antedating the period of their having obtained employment. There may be other cases where the employee has developed chronic otologic infections during the time of employment and, prompted by financial self gain, decides through cunning to misrepresent an injury to his previously infected ear. Such shams can be curtailed by proper Roentgenological studies, clinical observation, and the difference existing between the bones in question; hearing tests in conjunction with labyrinth studies may also be conducted.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, March 2, 1936.

In my practice I have found that injuries to the skull may cause deafness, but I have also experienced that it might be unilateral or bilateral, and, at times, no ear symptoms are presented at all; I have further learned that skull injuries, with or without skull fractures may have loss of hearing, either unilaterally or bilaterally. I have encountered such cases, on several occasions, at the New York University and Bellevue Hospital Otological Department. In the type of patient where there is a fracture of the skull and if it includes the petrous portion affecting the VIIth cranial nerve, one will find a complete loss of hearing, associated with marked vertigo, nausea, pallor, sweating and a paralysis of the accompanying VIIth nerve; this summarizes the essential differential points of diagnostic value in this condition. There may be either a total loss of hearing and a cessation of vestibular functions, or these conditions may not be affected at all by the trauma.

Feigned deafness is significant because of medicolegal relations to hearing disturbances preceding accidents, and symptoms of defective hearing may just be a pretense. Malingering is the conscious assumption of a disease picture, whereas hysteria is an unconscious assumption of such, and since no individual's innermost motives are ever of a single character both of these terms are invariably confused.

Neurasthenic deafness is rare. In this variety of cases a blowing tinnitus is present, and this sound is synchronous with pulse; also there is a mild vertigo present, which is evanescent. A labyrinth deafness may arise but it is fleeting in its essence. In this neurasthenic state, early in the examination of hearing, the individual responds well, but he tires very quickly as the examination is continued. Complete hysterical deafness may come on, generally instantaneously, following emotional imbalance. There is, however, absence of VIIIth cranial nerve pathological findings, and the cochleopalpebral reflex is manifested. Caloric and rotation tests produce nystagmus, and nerve reaction is created.

It is of the utmost significance then, that the ear specialist should be guarded in presenting his opinion. Expert opinion should be based upon various differential diagnostic points in conjunction with a thorough and carefully elicited history, laboratory studies, including X-ray plate examination, and a

Wassermann test. The utilization of a reliable malingering test must not be overlooked and, therefore, I present a special simple instrument which I devised for testing unilateral simulated deafness.

Procedure of Test: Let us assume that a patient claims a sustained injury to left side of head in the ear region and a resultant loss of hearing on the same side.

A new device for testing malingering in unilateral deafness. (A & B) Ear pieces for right and left ear (metal with rubber tips). (C) Rubber bulb for forcing air through rubber tube (D). (I) Rubber bulb for forcing air through rubber tubes (H & F). (J) For adjustment of air pressure to increase sound intensity. (G) Control tube for checking sound of air in ear piece (B). (E) Y-shaped tube connecting (F & H) tubes.

Method: The examiner places the complete instrument back of the patient so that he will be unaware of what is going on.

Step No. 1: A and B are placed in respective ears. The rubber bulb is compressed at C, and the sound of the air is transmitted through the rubber tube D, until it reaches earpiece at A. The patient is asked if he heard anything and he replies in the negative (since the left ear is the one he claims was injured, with subsequent loss of hearing).

Step No. 2: The rubber bulb is compressed at I by the examiner. The sound is transmitted through H and F, the air sound is then transmitted to B, where patient states he hears it. (G, the control, also transmits sound simultaneously as I is being compressed.)

Step No. 3: The earpieces (metal with rubber tips) are removed and the B side is placed on the left side and A is inserted on the right side. The same procedures are executed as in Step No. 1 and Step No. 2. This time, however, the bulb I is compressed first and, naturally, the patient will claim he does not hear it on the left side.

Step No. 4: A, earpiece, is then taken from the right ear and placed on right lobe; at the same time* G is closely brought up to the right ear without touching any part of the tissue; I is at the same time quickly compressed and the patient is asked if he heard any sound. If he makes a statement that he did not hear it, the examiner immediately knows that he is a malingerer, since he should hear with his good ear the sound emitted through B before it was reversed for final check up.

Advantages of This Test: 1. No human voice is necessary to conduct actual test. *2.* No tuning forks needed. *3.* No bone conduction is present. *4.* It is quickly and easily performed while the patient is kept guessing as to what the next step is going to be.

*Loss of hearing for higher tones of the musical scale is one of the most constant features related to the diseases of the labyrinth and the auditory nerve. In this case the note elicited by compression of rubber bulb I (is also sent through G), and is equivalent to value of 0.5 to 1.00 on Galton's whistle calibre).

807 St. Marks Avenue.

TONSIL EVERTOR. SEMISHARP FOR CUTTING OR TEARING; PILLAR RETRACTOR; KNOT TIER.*

DR. PHILIP S. STOUT, Philadelphia.

Tonsil eversion operations have been done by various methods and instruments. The writer has employed the eversion method of tonsillectomy in one way or another since 1919. During this time he has seen and used various instruments, many of which have been modifications of the Allis dissector, as is also the one shown here. One very well known instrument is the Lott evertor. It has a two-ended feature; one end for the

right tonsil and the other end for the left tonsil. In teaching this method to graduate medical students and internes, it has become more and more apparent that an instrument with only one end for eversion of both tonsils should be devised, since it is always a question of which end to use for that particular tonsil. If the wrong end is used it is difficult and the results may not be very satisfactory. This instrument answers that purpose; namely, it will evert both tonsils with the same end; also it has a semisharp cutting edge for tearing through the plica. The other end is a pillar retractor and knot tier. This instrument is made by Philadelphia Surgical Instrument Co., 1709 Sansom street, Philadelphia.

269 S. 19th Street.

---

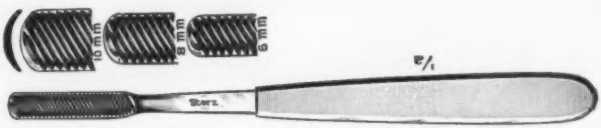
*Presented before the Philadelphia Laryngological Society, Feb. 4, 1936.
Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Feb. 19, 1936.

A NEW TYPE NASAL RASP IN PLASTIC SURGERY.

DR. SIDNEY ISRAEL, Houston, Tex.

For the purpose of rasping, planing or leveling a dorsal hump, spur or excess that may appear on the dorsum of the nose, one frequently resorts to the use of rasps. Heretofore, the rasps available for this purpose have been far from mechanically satisfactory, due either to the type or design of the rasping surface or the material from which they were made.

With this in mind and after careful thought, combined with a practical experience over a period of years, I have devised these rasps for consideration.



They are made of a specially tempered steel into three sizes, namely, 6, 8 and 10 mm. in width. They are curved in their long axes, are rounded and blunt at the distal end, and have a handle which fits firmly into one's grasp without turning.

A feature of these rasps, in addition to the above, is that the teeth are cut into the steel and run in an oblique direction. Because of the character of the teeth and the direction in which they travel, they promote an even, fast-cutting contact with which one is able to level off any hump or protuberance with the greatest of speed, surface smoothness, and evenness of the cut.

The cutting surface of the rasps is so made that they can be used only in a downward direction or on the down stroke. The curve in the long axes of the rasps permits the normal contour of the bone to be maintained, thus avoiding any flat

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, April 29, 1936.

or shelf-like appearance after their use. The type of the cutting surface and the oblique arrangement of the teeth permit a rapidity of execution in the accomplishment of the end results required, depending upon the contact made and pressure applied when the rasp is held in contact with the bone.

The three sizes make them widely applicable to any type of nose. They are easily cleaned and are mechanically an innovation when compared with previous rasps on the market. They are made by the Storz Instrument Co., St. Louis, Mo., whose advertisement appears on page 3 in this issue.

2010 Niels Esperson Building.

IN MEMORIAM

ARTHUR BALDWIN DUEL, M.D.

1870-1936.

Dr. Arthur B. Duel, another of the Old Guard in Otology, passed away at his country home at Laurelwood, near Pawling, N. Y., April 11. He was born in Granville, N. Y., Dec. 14, 1870, the son of Hiram D. and Almera J. (Hicks) Duel. He attended the University of Vermont and was graduated from Harvard, in 1894, with an M.D. Degree cum laude. In 1895, he began the practice of otolaryngology as Clinical Assistant in the Throat Department of the Manhattan Eye, Ear, Nose and Throat Hospital; was promoted, in 1897, to Assistant Surgeon and in 1902 became Surgeon of the Ear Department; in 1924, was Surgeon Director of this institution and in 1925, was made Chairman of the Board of Surgical Directors.

From 1896 until 1900, he was connected with the New York Eye and Ear Infirmary; from 1908 to 1913, he was Professor of Otology at the New York Polyclinic Medical School and Hospital, and Consultant Aural Surgeon at the New York Skin and Cancer, Babies and New York Board of Health Hospitals and at the Englewood Hospital, Englewood, N. J.

He was an active member of the American Laryngological, Rhinological and Otological Society, the American Otological Society, Inc., serving as a member of the Council of the former Society for a number of years, and in 1927 was honored with the presidency of the American Otological Society, Inc. He was Chairman of the Building Committee of the New York Academy of Medicine when the imposing new building at Fifth avenue and 103rd street was erected.

As Chairman of the Committee on Otosclerosis of the American Otological Society, Inc., he was an active factor in the work of this special committee until the time of his death.

During the World War he directed the work of the committee which organized the volunteer examination of 2,500 candidates for the Aviation Service.

While his contributions to the otologic literature were not frequent they were always authoritative and of high standard. Perhaps his most important contribution to otological science was his co-operation with the late Sir Charles Ballance in devising new methods for the surgical correction of facial paralysis by autoplastic nerve graft. The research work in this field included 200 operations on various types of anthropoid apes and cats to perfect the surgical technique and to observe the end-results of this autoplastic nerve surgery before it was applied to the human. More than 100 such operations have since been successfully performed on humans and the results published in the *Archives of Otology*, in 1932, and THE LARYNGOSCOPE, in 1934.

His was a rare personality, combining seriousness of mind, brevity of address, directness of purpose and, withal, a quiet demeanor and gentleness that won for him the esteem and respect of his colleagues and friends.

Some years ago Dr. Duel developed a flair for collecting Japanese prints and he had acquired one of the most important collections in the country of this phase of graphic art. To those of us interested with him in these hobbies he was like an enthusiastic child when displaying his choice and rare examples of Japanese color prints.

He was a Knight Commander of the Crown of Roumania, a member of the Calumet, Racket and Tennis, Harvard and Grolier Clubs.

In 1907, he was married to Miss Mary Kay Crawford. They had two children, Mary Hobart Duel and Arthur B. Duel, Jr. We who knew Dr. Duel as a devoted husband and father offer our sincere sympathy to his bereaved family and feel that in the passing of this otological colleague American otology has lost an active advocate who was always striving for higher standards and better work. M. A. G.

PROF. ROBERT BARANY.

After a long illness, Prof. Robert Bárány, of Upsala, Sweden, died April 8. With him Otology has lost one of its most significant figures. To Bárány we owe one entire chapter of otoneurology, that of diagnosis of labyrinthine disturbances; he not only created the methods of observation and analysis but, what is more important, he suggested ideas and advanced theories which during decades have stimulated research on the function and pathology of the labyrinth and its centres. This has not been generally realized, but scanning the literature on the labyrinth of the past 30 years one notes how often the authors, whether they expressed themselves for or against Bárány's assumptions, were working along the lead given by Bárány.

Bárány's best known accomplishment was the discovery of the caloric test. Important as it was to determine the existence of a constant reaction, it was much more important to give it theoretical explanation, because a diagnostic method is valuable only when well understood. Bárány's original theory of the production of an endolymph current by the change of temperature of the walls of the semicircular canals is today an established fact.

After the first papers in the *Monatsschrift für Ohrenheilkunde* in 1906-1907, Bárány's publications soon reached 200 in number. Most of them are well known to otologists; only the work of the last years is still but little known.

It is an important work. Bárány was trying to coordinate the fragmentary data, which constitute today's knowledge of anatomy and physiology of the nervous system, in order to explain the physiological phenomena in terms of the fine structure of the centres and of the properties of nerve cells and fibres. He knew that such a task could not at present be accomplished, but he also knew that no work would be finished unless carefully planned, and that neuro-physiological and psychological research are very much in need of new and

leading ideas. Bárány published but few of his results. Well known is his explanation of the bipartition of the granular layer of the optical cortex in mammals having binocular vision; and it will not be long until his explanation of the mechanism of hearing and his interpretation of the function of the closed chains of neurones of the brain become general in application.

The death of Robert Bárány is an irreparable loss.

R. L. DE NO.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

SECTION OF OTO-LARYNGOLOGY.

Meeting of March 13, 1936.

DR. C. L. OPPEGAARD read a paper entitled "Streptococcic Meningitis of Otitic Origin" and reported six cases, as follows:

Case 1. The patient, a male child, age 6 years, was a normally healthy child until the present illness. On April 9, 1935, he had a slight cold with a temperature of 100°. The next day he complained of pain in the left ear with a temperature of 101° in the morning. It was normal in the evening when the writer first examined the patient, at which time his ear had also ceased paining him. Previously another member of this Clinic had examined him from the general standpoint and found everything negative except for his present illness, which he pronounced a mild influenzal attack. The membrana tympani of the left ear as a whole had a dull appearance, but along the malleus and Shrapnell's membrane there was some congestion which, with a stretch of the imagination, one could say was increased over the normal. There was no tenderness over the mastoid area. The right ear, the nose, and the throat were negative to inspection. In view of these findings, his condition was not considered serious, but was felt to be the same as the many others otologists all see, which clear up with ordinary procedures. About 7 o'clock the next morning, the patient suddenly became stuporous, and was taken to the hospital in this semicomatose state. Even in this short space of time the manifestations and signs of intracranial involvement were very evident. Myringotomy of the left ear was done, and only a scanty bloody discharge was obtained. Within an hour the incision of the tympanic membrane had sealed together. He was somewhat rational the second day in the hospital. A beginning papillitis was seen on the third day and at this time he lapsed into complete coma. He gradually became more toxic and died on the sixth day following his mild onset.

Treatment consisted of repeated spinal drainage with injections of meningococcic serum, both intraspinally and intramuscularly. There was no evidence of mastoid involvement clinically and, due to his condition, an X-ray of the mastoids was not taken; and certainly their interpretation would not have changed the course of the treatment any, due to the apoplectiform onset of symptoms. He had had no previous attacks of earache. (Laboratory findings and spinal fluid findings given together in complete paper, after case reports.) Upon the interpretation of these rested the type of meningitis dealt with and upon the history and physical findings in each case rested the suggestion as to the mode of invasion of the cranial cavity.

Case 2. This patient, a male youth, age 13 years, was first seen at the hospital to which he had been transferred from his neighboring home town. He was in a semicomatose state and looked acutely ill. The following history was obtained: Two weeks previously he had developed an otitis media in the right ear, which had ruptured spontaneously, draining for about one week. Four or five days after the discharge stopped he complained of pain and stiffness in the back of the head and neck. He rapidly became worse and was brought into the hospital when his local doctor noticed that he was becoming stuporous. The examination revealed the classical signs of intracranial involvement except for negative eye grounds. The tympanic membrane of the infected right ear

showed a healed perforation with no other signs of inflammatory involvement. The left ear was negative. An X-ray examination showed no involvement of either mastoid. His progress to fatal termination was rapid, becoming irrational on the second day and expiring on the third.

Case 3: A male child, age 8 years, was first seen on March 8, 1935. At that time he had had a discharge from the right ear for two weeks, which had followed an illness of measles. The discharge had been very profuse and even with this drainage the ear had continued to pain. The patient looked and was acutely ill. The findings were typical of a right mastoiditis and a simple mastoidectomy was performed the same day. The operation revealed a well localized involvement of the whole mastoid area, which was thoroughly exenterated. He stayed in the hospital 17 days, with a moderately stormy convalescence, punctuated chiefly by attacks of pain like those that usually accompany an involvement of the petrous pyramid. However, he was discharged on March 25, with normal temperature and with the wound practically healed. He returned to the Clinic on April 8 with the history that for the last few days he had had pain in the right eye. There had been an increase of discharge from the middle ear. On the day the writer examined him, however, he was feeling quite well again. with very little drainage evident. We had in mind, of course, that here was a case of suppuration of the petrous portion of the temporal bone, but, against our judgment, were persuaded to let him return home. On April 12, four days later, he was brought directly to the hospital with the additional history that about two days after his last visit to the clinic he started to get listless and complained of pain in his left ear. This soon developed into a severe headache, radiating into his left eye. He complained no further about his operated (right) side of the head. Examination left no doubt that intracranial complications were present. The mastoid wound was healed. There was a slight discharge from the middle ear. In fact, all symptoms were now referable to the left side. There were no clinical signs of mastoiditis. An X-ray was not taken, for it was felt now that the right ear infection was the major cause of this new development, although later contacts have taught us to evaluate more definitely what we at that time thought were minor changes from the normal. The patient's condition rapidly became worse. Repeated spinal drainage was done, but he died a typical meningitic death on the fourth day. As an autopsy was denied, this case will always remain a question as to whether the extension was from the previously operated ear, or whether it was from the new focus in the left ear. The spinal fluids obtained were identical with those of the other cases, and that finding can be taken for what it is worth.

Case 4: The patient, a male child, age 7 years, on Feb. 26, 1935, developed a pain in the left ear. He was put to bed by his parents and seemed better the next day. The third day of his illness, however, he became worse and was seen by his doctor, who advised the parents that everything seemed negative except the ear complaint, and therefore advised them to consult an otologist. The father did not immediately follow this advice. The condition seemed stationary for the next few days, when suddenly on the sixth day of his illness, the patient had a convulsion and immediately lapsed into coma. He was taken to the hospital where the writer saw him for the first time in consultation with another member of our group. The patient was unconscious, pupils dilated, rigidity of the neck and back, with hyperactive reflexes. The left ear showed a highly congested ear drum with no bulging; the right ear was negative. The eye grounds were also negative. A myringotomy was performed on the left tympanic membrane, and a scanty bloody discharge obtained. The patient lived but four hours after entering the hospital, so no further procedures along diagnostic lines could be performed. His history did reveal that he had had earaches frequently in the past.

Case 5: This patient was a female child, age 6 years. In the middle of February she had a severe cold with bronchitis, from which she completely recovered. About two weeks ago she was taken suddenly ill with earache and the following morning after this attack her mother noticed that both ears had discharged some. Since that day there had been no further drainage. About

one week ago, the left side of her face seemed paralyzed, but the local doctor did not seem to be unduly alarmed until in the last 24 hours before entering our hospital, on April 23, 1935, when the patient started to become stuporous. Examination showed partial facial paralysis; left pupil dilated 50 per cent; and the ophthalmoscopic examination showed increased vascularity. There was a lateral deviation of the left eye, showing IIIrd nerve involvement. The tympanic membrane of the left ear was intact, with a more dull appearance than normal. The right ear was negative. There was some apparent tenderness over the left mastoid area. The general condition was negative, except for greatly increased activity of the reflexes. X-ray examination showed involvement of the antral area of the left mastoid. The parents were given a hopeless prognosis, but upon their insistence, we performed a simple mastoidectomy on the left ear. Macroscopically, the mastoid was negative, with questionable involvement around the antrum. Shie died on the fourth day of meningitis.

Case 6: (This case was under another doctor's care, who kindly gave the writer the information for use in this series of cases.)

The patient was a female, age 45 years. Two weeks before admittance to the hospital, she developed measles. Four days later she had an earache in the right ear and soon after the drum membrane ruptured spontaneously. There was a slight serous discharge for about one day. There was no further pain in either ear. Ten days after the onset of the measles, she began to feel nauseated, had occipital headache and sore throat. On the day of her admission to the hospital her temperature had been around 99.6°, but later in the day, following a chill, it rose to 104°. She immediately became delirious and vomited all food eaten that day. She later became slightly rational again and complained bitterly of photophobia, severe occipital headache and pains in the back. She exhibited marked rigidity of the neck with exaggerated reflexes. Eye grounds were negative. The examination of the ears revealed the left ear to be negative and, while there was no drainage from the right, the right drum membrane showed residual evidence of inflammation. An X-ray of the mastoids was negative. She became comatose on the second day after admittance and died on the third.

These cases reported with 100 per cent mortality occurred in the midst of an epidemic of mastoid infections in which proper operative treatment effected 100 per cent cures. These isolated cases seemed to arise from the same etiological causes, but rapidly assumed a different complexion, with fatal termination as shown in the case reports. In an attempt to analyze the route of invasion to the brain, the following factors were considered: 1. Increased virulence of the invading organism; 2. preformed pathways; 3. presence of "silent mastoiditis;" 4. invasion by way of the vascular channels. In the spinal fluids of all these fatal cases, the non-hemolytic streptococcus was isolated. In the final analysis, it was felt that the mechanism of invasion came about by the entrance into the blood stream of minute particles from the thrombi in the vessels of the middle ear or areas contiguous to it.

DISCUSSION.

DR. J. A. WATSON said that his own experience corresponds to that of Dr. Oppegaard in this way:

These cases reported with 100 per cent mortality occurred in the midst of an case of streptococcic meningitis get well and he had seen many of them. He thought that one or two of the men here have had cases that recovered and he would not say that such a thing is impossible. He believed, however, that the question of operating in a well-established case is hardly to be considered. Last summer he saw a case in a boy, age about 10 years, in the northern part of the state. He learned from the history that the boy had had a suppurating ear the previous summer with some symptoms of mastoid trouble, but nothing was done about it. Evidently that was the time to have operated. When Dr. Watson saw the case, he considered it perfectly hopeless. There was a very great

increase in the cell count in the spinal fluid. The boy's parents lived in Chicago, and when Dr. Watson telephoned them and told them of the boy's condition, they told him to operate if he thought it wise, which he did not. The boy was sent by plane to Rochester, where he was operated and died the following day.

Only a few days ago Dr. Watson received a phone call from the country that a patient was being sent in and to be ready to operate as soon as he arrived. The man, of middle age, had had severe pain in the ear, which had been discharging for some time. He had suddenly became comatose. The spinal puncture proved that the condition was streptococcic meningitis. The man died about an hour after arriving. The mortality in all such cases is practically 100 per cent.

Dr. V. J. Schwartz felt that Dr. Watson's point that not 1 per cent of these patients get well, is entirely true. As a matter of fact, all the reported recoveries from streptococcic meningitis have been carefully collected, and they now number 60 cases. Dr. Schwartz had previously reported one such case from the Minneapolis General Hospital. This patient had a streptococcic meningitis with a polymorphonuclear count in the spinal fluid of about 4,000 and a bilateral acute mastoiditis. The patient recovered. He said he did not know which portion of the treatment was of the greater consequence, the double mastoidectomy or the flushing of the spinal canal twice a day with a solution of magnesium sulphate. In all probability both factors were indispensable.

Dr. Schwartz felt that Dr. Oppegaard is to be commended for the lucid and courageous presentation of his cases. Unfortunately, not all men present their bad results so willingly, and yet these cases often are much more instructive than those in which the results are good. A group of articles appeared recently in the form of a symposium on the subject of streptococcic meningitis by members of the New York City Health Department (Eggeston, et al., Annals Otol., Rhinol., Laryngol., Sept., 1934). These articles were of much scientific as well as practical interest and any one interested in the subject would do well to read them. Of the total number of such cases, approximately 35 per cent also had a streptococcic bacteremia. It is interesting to speculate which came first, the meningitis or the bacteremia, as in the old question of the hen and the egg.

Dr. Carl Larsen said his only comment was to congratulate Dr. Oppegaard on his excellent presentation and a complete study of his cases. It is an unusual number of cases in so short a time. Dr. Larsen stated that if he had had them in his practice, he would have been very much discouraged. He felt that in the majority of these cases the method of spread is vascular. He also felt that Dr. Watson's estimate of 1 per cent recovery is very much too high. He would say the mortality in otologic meningitis is nearer 99.9 per cent.

Dr. Oppegaard said he had nothing special to add except to thank the members for their kind consideration of his paper.

Dr. D. L. Tilderquist reported two cases of "Foreign Bodies in the Nose." Specimens of the removed foreign bodies were shown.

These two cases are presented to bring to our attention the possibility of foreign bodies in the nose. In the case of children, one might expect at times to find buttons, wads of paper, peas and what not in the nose; but not so in the case of adults. A foreign body in the nose may not be readily seen. Both of these cases to be reported had been examined one or more times "elsewhere."

The symptoms of a foreign body in the nose, as you know, are the ordinary symptoms of nasal irritation, sneezing, obstruction, discomfort, etc. There is increased secretion; it is at first watery, but later it may become purulent and acquire a bad odor. If an ulceration occurs, the secretion is often blood-tinged. The foreign body might be a sequestrum or a rhinolith. The formation of the latter is analogous to that of a gallstone or a vesicle calculus. In the course of my practice I have seen a few rhinoliths.

The method of removal of a foreign body suggests itself, of course, in each case. The membranes are shrunk thoroughly with cocain and adrenalin and the foreign body grasped with a forcep or teased out with a blunt hook.

Case 1: This patient appeared at the office in July, 1935, complaining of a disagreeable odor and a general feeling of discomfort in the nose. These symptoms had been present for about two months and had appeared following an automobile accident in which he had been involved. At that time he was riding in a friend's car, sitting in the front seat with the driver, when his car bumped into a car ahead, which had stopped suddenly, and the patient was thrown against the windshield. He sustained a cut in the forehead, but otherwise received no injuries as far as he knew.

On examination there was found much secretion in the right nasal space with an odor resembling that of an atrophic rhinitis. After shrinking the membrane and removing the secretion, something black could be seen in the middle meatus. This was grasped with a forcep and a flat foreign body came out, which was about 1½ inches long and ¼ of an inch wide, and resembled a piece of hard rubber. It was undoubtedly a fragment of a strip which had held the windshield glass. The patient knew nothing of getting this into his nose.

On close inspection of the nose it was found that the anterior half of the attachment of the middle turbinate was divided and the anterior end hung loosely in the nose. Transillumination showed the right maxillary moderately dark.

He came back about New Year's day, complaining of a headache of two week's duration which centered over both temples. Thinking he might have a sinus infection, an X-ray film was taken. This showed definite haziness of the right maxillary, but did not give any suspicion of any other foreign body. Transillumination showed again a dark right maxillary. The cavity was irrigated through the natural opening, which was easy of access in this case, but the solution returned entirely clear.

It is, of course, entirely possible that another foreign body may be in the nose or in the right maxillary, but there was no way of determining this except possibly by exposing the cavity of the right maxillary. This was not done, as his symptoms subsided and he has not been seen since.

Case 2: This patient was seen at the office in August, 1935. For three years he had experienced discomfort in the left side of the nose, obstruction to breathing, increased secretion and occasionally the appearance of a little blood. There was no history as to the onset. He was otherwise in good health except for reduced vision, which was found to be due to a bilateral senile cataract.

On first inspection of the nose, nothing of interest presented itself. There was a sharp spur of the septum to the left, closing most of the space except a little slit at the floor. After anesthetizing the membrane with cocain solution, a probe was used and a hard obstruction encountered back of the spur which yielded a grating sensation when the probe touched it. Thinking there might be a sequestrum or a calcareous gathering, a slender instrument was pushed back and attempts made to hook the body forward, but on account of the spur, it proved all in vain. It was found that after each attempt the body lay farther back in the nose than before. This suggested the idea of pushing it through into the throat. Fearing the thing might fall directly into the open larynx, the man was instructed to fill his lungs, then keep on phonating while I pushed in the nose. All at once an astonished expression came over his face, he forgot to sing, and then calmly spit out something that gave a metallic ring when it hit the basin. We together curiously examined a little jagged stone-like body about ½ inch in diameter, which we agreed looked more like a cinder from a coal stove than anything else we had seen.

Then he remembered that about 25 years ago, in the bicycle era, he had on one occasion been riding a bicycle on a newly built path, had fallen off and

had pushed his face into a bank of loose cinders. After recovering from the bruising he had not noticed any further bad results, but now it seemed quite evident to him that the cinder must have entered his nose at that time.

He left for home, taking the cinder triumphantly with him, but with the agreement to send it back to me later, which he did.

Dr. George C. Dittman said there is not much to discuss about this paper except to report a similar case he had had. The patient was a man, age about 35 years, who came to the office with a discharge from the left side of the nose and a discharge from the ear on the same side. Dr. Dittman suggested a sinus operation and the man finally consented to have it done, and came in the next morning. In preparing the nose for operation, Dr. Dittman said he saw in the posterior third of the nasal passage a grey colored obstruction which he thought might be a spur. Believing that he might break the spur off with forceps he applied force and pulled out a cherry stone, which was found encrusted with a calcareous deposit. The patient could not remember getting it into the nose, but he evidently had had it the greater part of his life, and also the suppurating ear.

Dr. V. J. Schwartz reported a case of congenital cyst of the larynx in a newborn.

This was so large as to cause asphyxia, and while the patient was temporarily revived by tracheotomy, the infant was too weak to survive more than 12 hours. The cyst was about 2½ cm. wide and 5 cm. long and in the left lateral wall of the hypopharynx, compressing the trachea and larynx almost to closure. The origin of this cyst is not clear. It may have been of bronchial development, but it is more likely to have been of the mucous type. Specimen shown.

Dr. Anderson Hilding reported two cases: a. Labyrinthitis, meningitis, late facial paralysis with recovery; b. Mastoiditis complicated by dislocation of the axis. Lantern slides of both cases were shown.

NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTO-LARYNGOLOGY.

Meeting of Jan. 15, 1936.

Laryngeal Conditions, Case Report. Dr. Jno. J. Madden.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Parapharyngeal Infections: 1. Chronic sialadenitis (submaxillary); 2. Abscess of submaxillary gland with calculus; 3. Retropharyngeal cellulitis-tracheotomy; 4. Acute tonsillitis, retropharyngeal abscess, parotid gland abscess. Dr. Earl F. Baker, New York City.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Management of Face Injuries: 1. Fracture of superior maxilla (infra-orbital bones); 2. Fracture of nasal bones, fracture of superior maxilla, laceration of nose and face, fracture of clavicle, laceration of knee; 3. Resume of cases of fractured nasal bones. Dr. Frederick E. Dempsey, New York City.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Mastoid Conditions. Dr. Raymond J. Gaffney.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Esophageal Conditions. Dr. Anthony Nigro.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Malignancies of Ear, Nose and Throat. Dr. Edward Markey Pullen.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. GERALD H. COX: I will make just a few remarks about this subject of fractures of the nose and fractures of the bones of the face. I was very much interested in Dr. Dempsey's paper in regard to the treatment of fractures of the nose, that is, reduction of the fractures. It seems to me that the two important points are, in the first place, that you must thoroughly and absolutely reduce the fracture. If the nose has been shoved way over to one side, push it back to the midline and be sure the fragments are thoroughly reduced. If you do that, in a great many cases no splinting is necessary. If a splint is needed, on the Bellevue service we have had the best luck with ordinary copper sheeting material which the tinsmiths use on roofs. It is malleable and you can cut out a little piece and mould it to the nose, put it across the nose with a strap across it and overcorrect, if necessary. I was interested to hear Dr. Dempsey speak of using Blair's method of a suture through the tip which is anchored to the periosteum. I did not know that this method had been used for the treatment of recent fractures. I thought it was applicable in doing plastic surgery of the tip of the nose.

Fractures of the malar and zygomatic arch are not common. If one gets one of those cases, for the first time, it is interesting to go around and see what advice one gets. When I first had one of them, I remember an orthopedist told me that he made a little incision over the malar bone and used a corkscrew to pull the fragment out. Another man used a hook and pried out the malar bone with that. The first case I had was a bad fracture of the malar zygomatic arch. I took a periosteal elevator, made an incision in the mouth through the mucous membrane, went under the malar bone and pushed it out. I got a very nice result and needed no splint. I subsequently went to the morgue and with a mallet produced a fracture of the malar bone and tried this procedure. I found that not only fractures of the malar bone could be reduced by this method, but also fractures of the malar zygomatic arch well back toward the articulation with the temporal bone.

DR. DAVID H. JONES: I was very glad to have Dr. Madden emphasize the feature about hoarseness. We find that so many men in general practice, when a patient comes to them over the age of 50 years complaining of hoarseness, treat them for months, and do not even look down the larynx. Another point is that many men practicing oto-laryngology do not know themselves how to look down into the larynx. We find that many of the younger men do not know how. I cannot impress enough on you that when you have a case of hoarseness, do not just treat it for chronic laryngitis. There is some cause there. At the last meeting in Toronto Dr. Jackson made the statement that hoarseness in a person over 40 years should be called carcinoma until it is proven to be a non-malignant case.

In regard to making a diagnosis when you do a biopsy, if you have a single biopsy and the report is negative, do not rely on that if the case looks to you clinically as if it were a new growth. Repeat the biopsy. We had a case today which I have been watching for four years. Clinically, it was a new growth. We had biopsies and we had several instruments made in order to get what we thought was a good biopsy. They were all negative. Consequently, Dr. Allen went ahead and did a laryngofissure. We were surprised to find on removing the growth that it was carcinoma. That man has had 34 deep X-ray treatments. Another point which Dr. Madden made was that in nearly every case hyperkeratotic papilloma will become carcinoma.

In Dr. Nigro's paper, in regard to the meat, Dr. Jackson makes this point: that in all cases where meat is occluded in the oesophagus, there is some cause for it.

Regional Anesthesia in Ear, Nose and Throat Surgery. Dr. John M. Lore.
(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. HENRY M. SCHEER: I would like to say just a few words. I cannot pass up this opportunity to compliment the chairman and the members for permitting Johnny's gang to come up here and present as they have. Knowing John Lore for 25 years as I have, from the first day we entered college, I can safely say that any time you see his name on a program to present a subject, particularly if it be an informal talk, do not miss coming, because you are going to get many points that are not in the books and many points that are of more value for practical experience. I think the gang has someone to be proud of in their chief. There is no question about that. If you have been present at any of his talks or presentations, you have found that out before now.

I have just a few remarks to make in regard to local anesthesia. I think I have applied local anesthesia in my work in almost every procedure spoken of by Dr. Loré except the external frontal operation and the larynx work. I can speak particularly of the mastoidectomy done under local anesthesia. A little later in the spring the chairman has been kind enough to let me present a series of cases of sinus thrombosis, among them a man, age 69 years, on whom

I did a mastoidectomy, blocking of the lateral sinus and tying off of the internal jugular under local anesthesia. Do not carry away the impression that local anesthesia is not entirely satisfactory in adults. I think it is just as safe and, if properly applied, just as nice a procedure to use as in a child, with only morphine and some hyoscine as preliminary preparation. Just yesterday I had a patient, age 26 years, who received ¼ gr. of morphine sulphate and 1/100 gr. of hyoscine one hour before operation, and another 1/16 gr. of morphine sulphate one-half hour before operation. The patient was practically sound asleep at operation. I will say that during the procedure the mallet is not used. The tip is removed first with the rongeur and the mastoid cortex removed completely and the conventional procedure done with the rongeur, then the exenteration done in the usual way. Therefore, even in aduls where one does not wish to use the mallet, one can avoid doing so by applying that procedure.

I will take this opportunity again to compliment Dr. Löré on his very excellent work. He is very modest and humble about his work, but I know that he knows his business and he is right on his toes.

Dr. Julius I. Klepper: It is very appropriate to compliment Dr. Loré on his paper, but I want to point out that in discussing anesthesia of the mastoid, it must not be forgotten that the mastoid bone itself is not supplied by the great auricular nerve. It is supplied by the recurrent meningeal or nervus spinosum and if you do a mastoidectomy under local anesthesia, you do it because the bone is not as sensitive as the skin. The bone itself is not supplied by the great auricular nerve. It is the recurrent meningeal or nervus spinosum which supplies the bone. The great auricular in conjunction with the small occipital do supply the integument over the mastoid and posterior part of the auricle.

NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTO-LARYNGOLOGY.

Meeting of Feb. 19, 1936.

Malignancies of the Nasopharynx. Dr. Louis Kleinfeld.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Unusually Early Pleuropulmonary Suppuration in Sinus Thrombosis. Dr. Harry Rosewasser.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Gradenigro's Syndrome Simulating Expanding Intracranial Lesion. Dr. Walter L. Horn.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Reparative Processes in the Labyrinth Secondary to Meningococcus Meningitis. Dr. Joseph G. Druss.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Thrombophlebitis of the Jugular Bulb and Lateral Sinus of Non-Otitic Origin; Important Ear Findings. Dr. Samuel Rosen.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Thrombophlebitis of the Jugular Bulb and Lateral Sinus of Non-Otitic Origin; Important Ear Findings. Dr. Irving B. Goldman.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Thrombophlebitis of the Jugular Bulb and Lateral Sinus of Non-Otitic Origin; Important Ear Findings. Dr. J. L. Maybaum.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Sphenoiditis with Meningitic Symptoms. Dr. Rudolph Kramer and Max L. Som.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Complications of Acute Sinusitis With Special Reference to Bacteremias. Dr. Joseph L. Goldman.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Thrombophlebitis of the Jugular Bulb and Lateral Sinus of Non-Otitic Origin; Important Ear Findings. Dr. Samuel Karelitz.

DISCUSSION.

Since the purpose of the preceding papers is to make known a clinical picture, which hitherto has been recorded in the literature only as a postmortem finding, I presume some repetition is justifiable.

First, I should like to trace the observations which led to the correlation of the otitic finding and the clinical diagnosis of retrograde jugular bulb and lateral sinus thrombosis in the second and third cases referred to by Dr. Rosen.

During the course of illness of Case 1 (the child who had left-sided mastoiditis and sinus thrombosis), the patient suddenly developed an unexplained swelling of the right cervical glands. This caused me to look into the right ear. To my surprise, I saw a peculiar bluish discoloration of the posterior inferior portion of the right drum. In the subsequent 10 days this otitic discoloration increased and was not associated with disturbances such as pain or alteration of hearing or canal wall changes. Because the color suggested passive congestion, I inquired as to the circulation of the tympanic membrane from Dr. I. Friesner. When I was told that it drained into the bulb, I asked whether it was conceivable that this child might have thrombosis of the right jugular bulb and lateral sinus to account for this drum engorgement. I also mentioned to Dr. Friesner that I had never experienced observing a drum change which lasted so long and was so quiescent or silent. Surgical interference of the lateral sinus was not considered beyond mentioning that the finding was hardly enough justification for surgery. The finding of thrombosis of the right jugular bulb and lateral sinus at postmortem examination suggested very strongly that the suspected relationship between the otitic discoloration and bulb and sinus thrombophlebitis were warranted.

An opportunity to check the validity of these findings came in the second case described. After the ocular complication had been taken care of, the child seemed quite well for almost a week, after which the temperature rose to 103°. The only explanation which could be found for this fever was a marked congestion of the right tympanic membrane. The drum was dusky red and this change involved the adjacent canal wall. It will be recalled that a slight pink color of the right drum was first noted simultaneously with the appearance of the right ocular chemosis and that this change, which was then regarded as being a catarrhal inflammation of the drum, progressively increased without ever causing pain or disturbance of hearing.

A myringotomy was done, but only blood was obtained. It was then that I stated that the only time I had ever seen such a peculiar involvement of an ear drum was in a case (Case 1), which proved to have lateral sinus and bulb thrombosis, which I felt was also present here (Case 2).

Forty-eight hours later a rise in temperature to 105°, a chill and the finding of streptococci in the blood stream were sufficient to make me feel quite certain of my diagnosis. Nevertheless, it would have been very difficult to insist on operative interference through what appeared to be a normal mastoid, on the experience of only one case. Roentgen examination of the mastoid erroneously indicated breaking down of the bone. At operation no changes of the mastoid were noted, but the sinus and bulb were found to have been occluded by thrombosis and the phlebitis extended to within a few centimeters from the torcular. This child had an uneventful postoperative recovery. The ear finding was a definite aid in this case in that it suggested sinus thrombosis, for even at operation, several otologists felt that the lateral sinus should not be explored. Had we followed this advice, the most important step of treatment might have been passed up.

After the experiences with the first two cases, I was fully convinced of a relationship between the otitic finding and the sinus thrombosis and also that retrograde bulb and lateral sinus thrombosis spreading from internal jugular thrombophlebitis was a clinical entity which was recognizable. I, therefore, wrote a paper pointing out these findings and asked Dr. Rosen to proofread it.

Shortly thereafter, Dr. Rosen overheard the early part of the history of Case 3, and promptly recognized the similarity of this case to our second one. Dr. Rosen asked for permission to see the ear of Case 3. Upon finding engorgement of the ear drum on the side corresponding to the neck infection, he suggested that the sepsis probably was due to sinus thrombophlebitis and recommended immediate surgical interference. Dr. Maybaum, with whom I had discussed my first two cases some time before, examined Case 3 and independently arrived at the same conclusion, namely, that operation for sinus thrombosis was indicated. The operation was done after considerable delay and the findings were identical with those of the first two cases.

Since submitting a report (November, 1935) on the three cases here referred to for publication in The American Journal of Diseases of Children, I have discussed the matter with many physicians and have found several more cases which fall into the same group. Thus Dr. A. Kaplan, one of our neurosurgeons, to whom I had described my cases, was subsequently asked to see a child who, after an acute pharyngitis, developed neck suppuration with septicemia and cerebral symptoms. In view of the proximity of the neck abscess to the internal jugular vein and cerebral symptoms, he suggested lateral sinus thrombosis and recommended operation. The sinus thrombosis was found as was a perisinus abscess. The child made an uneventful recovery. The mastoid was normal. The ear drum on the same side of the neck abscess was slightly discolored. This fact was disregarded, yet I believe that it was probably similar to the finding which I have described.

Upon describing my findings to Dr. Alfred Braun, he promptly told of a case almost identical to our third case. His patient, a child, developed a neck abscess after an acute pharyngitis. On incision into the abscess he found that there was an internal jugular phlebitis. This child developed a septicemia which Dr. Braun interpreted as being due to a retrograde thrombophlebitis of the bulb. He found bulb and lateral sinus thrombosis at operation and the child recovered. This child developed otitis media after the internal jugular phlebitis was first seen. The mastoid was normal at operation.

Dr. J. Pincus, in discussion of a paper which I read on this subject before the New York Pediatric Club in November, 1935, commented that one of three cases of internal jugular thrombosis, which he had seen at the Brooklyn Jewish Hospital, developed thrombosis of the bulb and lateral sinus, which was cured by operation. He did not recall the otitic picture but assured me that if there was any otitic involvement at all, it was slight and no mastoiditis had been found at operation.

After reading my paper, Dr. Friesner likewise recalled a patient who developed lateral sinus thrombosis after cavernous sinus involvement. This man had a peculiar otitic congestion, which Dr. Friesner now believes must have been comparable to the findings which we have reported. Dr. Friesner will probably refer to this case in his discussion.

Our findings suggest very strongly to me, as they do to Dr. Goldman, that the so-called cases of primary jugular thrombosis are in some instances similar to those just described. This impression on my part differs from the opinion expressed by Dr. Maybaum; still, I believe that since some of the cases of primary jugular thrombosis had only very slight otitic manifestations, it is quite possible that they were merely congestive phenomena as described in my cases of retrograde or non-otitic sinus thrombosis.

Finally, returning to the ear drum manifestations which we described, we can say only this: They were present in our three cases, in the case seen by Dr. Kaplan and the one recalled by Dr. Friesner. In our three, it was a definite aid in establishing a diagnosis. I appreciate, of course, that the otitic changes, if present at all, may vary and may not be constant, since it is unlikely that the circulation is similar in all cases. I feel, however, that when the ear changes are present along with sepsis, especially in pharyngeal and neck suppurations, as was emphasized by Dr. Maybaum, retrograde jugular bulb and lateral sinus thrombosis must be thought of and ruled out.

Even if the otitic findings should fail us in future, their presence in the reported cases will have been helpful, since they served to focus our attention on the clinical picture of retrograde jugular bulb and lateral sinus thrombosis of non-otitic origin.

GENERAL DISCUSSION.

DR. ISADORE FRIESNER: I am sure there are so many points that have been raised this evening that you would like to discuss that I am very loath to waste any of your time through anything that I have to say. I want to call attention, however, to just one or two points in passing. The most marked ear change in this condition that I have ever seen I observed in a doctor many years ago who, following a tonsillitis, had a cavernous sinus thrombosis. His ear gave the appearance of being edematous and red. There was so evidently a venous congestion in the ear, the manubrial plexus and that about the annulus standing out so prominently in the thickened drum, that there was no question in my mind as to the pathogenicity of this phenomenon. Furthermore, there was in this case a Griesinger's sign, the typical edema about the emissary vein. I was asked to see him when he was moribund, with the suggestion that probably this whole condition was due to an undiagnosed otitis. However, here, as in some of the cases of so-called primary bulb thrombosis that have been reported from my service at Mt. Sinai Hospital, there was no history and no evidence of an otitis. As Dr. Maybaum has said, in all of the cases that we have reported as primary bulb thrombosis there was either a frank suppuration, that is, a perforative otitis, or an extremely suggestive history of pain in the ear for one or more days. The characteristic thing so far as the ear is concerned in these cases is that there was no otitis.

My feeling in the matter at present, from what clinical experience we have had, is as follows: If there were a sepsis of pharyngeal origin with a thrombophlebitis of the jugular, and if the juguar were explored and there were no bleeding from the bulbar end, that is, the upper end of the vein, and there were these characteristic ear findings, I believe I would want the lateral sinus explored immediately at that time because these cases have a peculiarly virulent tendency to spread to the cranial venous sinuses. Now, in the second group of cases, if the vein has already been operated and the sepsis persists and the upper end of the vein on the secondary exploration is not patent but is thrombosed, I would want the lateral sinus explored, whether there were ear findings or not, because in one of the cases that I saw, the ear findings were extremely evanescent and what may be there one day may be gone two or three days following. I think the important thing — and incidentally, this is just as much a surprise to me as I hope it is to many of you — is that there is a distinct group of cases of jugular phlebitis following throat infections in which the progress of the disease is almost solely retrograde, to me a most unusual condition; but recognizing the fact that such a condition may exist and also aware of its extreme virulence as evidenced by its rapid spread, I think if we are to see any considerable proportion of these cases saved, we must operate early and completely.